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Association.

TRANSACTIONS
OF THE
AMERICAN GASTRO-
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ASSOCIATION
1919

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TWENTY-SECOND ANNUAL MEETING

OF THE

AMERICAN
GASTRO-ENTEROLOGICAL
ASSOCIATION

HELD AT THE

HOTEL TRAYMORE, ATLANTIC CITY, N. J.

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CONSTITUTION AND BY-LAWS
OF THE
AMERICAN GASTRO-ENTEROLOGICAL
ASSOCIATION.

CONSTITUTION.

ARTICLE 1. This Association shall be known as the American Gastro-Enterological Association.

ARTICLE 2. The object of this Association shall be the promotion of investigation of the normal and pathological conditions of the digestive organs.

ARTICLE 3. Membership in this Association shall be of three kinds: (a) Honorary, (b) Corresponding, (c) Active.

(a) Honorary membership shall be restricted to such as have attained an international reputation by their published works and have scientifically furthered the subject of gastro-enterology. Their nomination shall take place at the annual meeting, upon the written suggestion of at least six active members and on the recommendation of the Council of the Association at the next annual meeting. Their election shall be by unanimous vote of the members present.

(b) Corresponding membership shall be restricted to foreign scientists in recognition of meritorious work within the scope of the Association. Candidates for such membership shall be proposed by at least six active members and receive the recommendation of the Council at the next annual meeting. Their election shall be by unanimous vote of the members present.

(c) Active membership shall be restricted to American and Canadian investigators and practitioners who have published meritorious work in normal or pathological anatomy or physiology, in medicine or surgery of the digestive canal and its secretory appendages, and who enjoy an unimpeachable moral standing in the medical profession.

Names of candidates for active membership must be: (1) Proposed by two active members who are not members of the Council, such proposal to be submitted to the Committee on Admissions and Ethics, which should report to the Council as soon as possible; (2) Must be recommended by the Council and (3) must receive a majority of the votes of active members present at the annual meeting of the Association.

The election of active members shall take place at the annual meeting of the Association and shall be by ballot.

It shall be the duty of the Secretary to notify active members of all nominations that are to be submitted to the Association at the annual meeting.

ARTICLE 4. The officers of the Association shall be a President, two Vice-Presidents, a Secretary and Treasurer, the latter two offices being vested in one; also three specially chosen active members to be known as Councilors, who, together with the officers, shall constitute the Council of the Association. The said Councilors shall be elected as follows: one for one year, one for two years, and one for three years.

ARTICLE 5. There shall be a Committee on Admissions and Ethics. It shall consist of the President and the Secretary and three other members, not members of the Council. These members shall be elected at the annual meeting at which this Amendment shall be adopted for the following terms: one for three years, one for two years and one for one year. At each subsequent annual meeting one member shall be elected for the term of three years to fill the annual vacancy. No member of the Committee shall be eligible for re-election until after one year shall have elapsed from the end of his term of service. It shall be the duties of the Committee on Admissions and Ethics to examine the merits of a candidate, to investigate any charges made against a member, and to report the results of the examinations and investigations to the Council in writing in the shortest possible time. It shall also be the duty of this committee to study the active work in science and practice of gastro-enterology accomplished in this country and Canada and to invite the meritorious workers to join this Association.

ARTICLE 6. The election of officers, Councilors and of the members of the Committee on Admissions and Ethics shall take place at the annual meeting and shall be by ballot.

ARTICLE 7. Vacancies in the offices occurring in the interval between the annual sessions shall be filled temporarily by the Council.

ARTICLE 8. The annual meeting of the Association shall be held at a time and place to be decided by the Council.

ARTICLE 9. The President shall be the chairman of the Council.

ARTICLE 10. There shall be annually two meetings of the Council; one shall take place at the call of the President, shortly after the annual meeting of the Association; the other shall take place at least four weeks previous to the annual meeting. It shall be within the discretion of the President to conduct this latter meeting by correspondence.

ARTICLE 11. The Council shall manage the affairs of the Association in accordance with the Constitution and By-Laws, and its minutes shall be reported to the Association at the annual meeting.

ARTICLE 12. This Constitution may be amended by a two-thirds vote of all the active members at any annual meeting, provided that notice of proposed amendments has been given in writing to all the active members of the Association by the Secretary, and provided fur-

ther that such proposed amendments shall have been submitted to the Council and held over for one year.

ARTICLE 13. Any member failing to attend three consecutive annual meetings without an excuse acceptable to the Council shall be dropped from the roll. The Secretary, however, shall be required to call the attention of a delinquent member to the facts in his case previous to the third meeting.

ARTICLE 14. This Association adopts the Code of Ethics of the American Medical Association. Charges of advertising, of publishing knowingly false scientific statements, of actions unbecoming a gentleman and a high standing physician, etc., shall be investigated by the Committee on Admissions and Ethics. The accused shall have liberal opportunities to clear himself of the accusation. The Committee shall report its findings to the Council, and on the recommendation of this body an offending member may be expelled by a three-fourths vote of those present at the annual meeting.

BY-LAWS.

ARTICLE 1. The President and Vice-President shall discharge such duties as are implied by their respective offices. The President shall preside at all sessions of both Council and Association.

ARTICLE 2. The Secretary shall attend to the usual clerical duties of the Council and Association. As Treasurer, he shall keep a record of the payments and arrearages of dues, and report on both at the annual meeting.

ARTICLE 3. The Council shall report its transactions to the annual meeting of the Association. It shall superintend the publication of scientific papers, but shall not appropriate for such printing an amount exceeding the sum of \$100, without the vote of the Association. The order of business shall be arranged by the Council before the annual meeting. It shall pass upon the eligibility of candidates for membership.

ARTICLE 4. The yearly dues of active members shall be five dollars, payable in advance. Any member whose subscriptions shall be in arrears more than two years shall be reminded of the fact by the Treasurer, in writing in event that payment be not then made he may, on vote of the Council, be dropped from the roll of the Association. It shall be the duty of the Treasurer to report all members who are in arrears to the Council.

ARTICLE 5. When sufficient money is in the treasury the membership dues may be omitted when decided by the Council.

ARTICLE 6. These By-Laws may be amended, repealed or suspended by a two-thirds vote of the members present at any meeting of the Association.

ARTICLE 7. The quorum of the Association for the transaction of business, but not for the reading of papers, shall consist of seven active members.

ARTICLE 8. All titles of papers submitted for reading at the annual meeting shall be sent to the Secretary not later than four weeks before the date of the meeting.

ARTICLE 9. A typewritten abstract of not over three hundred words, of every paper to be read, must be sent to the Secretary at least four weeks before the annual meeting. It shall be the duty of the Secretary to send to each member of the Association a complete list of the papers to be read at the annual meeting, with their full titles, at least two weeks before the annual meeting.

ARTICLE 10. Papers submitted to be read before the Association shall not have been previously published or read elsewhere. Papers shall not exceed the limit of fifteen minutes without the consent of the majority of members attending.

PRESIDENT'S ADDRESS

WALTER A. BASTEDO,
NEW YORK.

GENTLEMEN:—In electing me to be your President you have bestowed upon me a very great honor, and for this I thank you. I deem myself unworthy of this distinction, and I should feel the filling of the office beyond my powers were it not for the high quality and interest of you members of this Association. Through your efforts and those of some invited guests we are at the commencement of a two-day intellectual treat. Do you realize how much is contributed to the profession by such gatherings as ours, where opinions are exchanged among a group of men who by studies of precision in a special field replace diffuseness with definiteness and cloudiness with clearness? Do you realize that to the profession at large you are the leaders in gastro-enterology, the men who make the daring and valuable advances which can only be made by those who, through limitation of their field of work, can concentrate their experiences and their thoughts? Along with this goes a great responsibility in the expression of opinion. Let us make every endeavor to keep the personnel of our membership of the highest quality.

In response to the requests of some of our members I am going to address you on the pharmacologic action of certain drugs used for stomach effects, and, owing to time limits I shall confine my remarks to atropine, pepsin, rennin, hydrochloric acid, bitters, cerium and bismuth.

POINTS IN THE PHARMACOLOGY OF CERTAIN DRUGS USED FOR STOMACH EFFECTS.

Atropine.—This drug has such a decided action on certain secretions and certain motor functions of the body that it has for long years been assumed to have similar effects on the stomach. Yet clinical beliefs that have been supported by the leaders in medicine have so often been forced aside when the light of experiment has been turned upon them, that it behooves us to appraise carefully the value of such a drug as atropine in its application to the stomach.

I. *Secretion*.—Observations on human subjects have been made by Crohn with the fractional method of gastric analysis. His results were: (1) *In hyperacidity cases with normal secretion period*.—One mg. (1/65 grain) of atropine sulphate administered to the patient hypodermatically three-fourths hour after the meal, had little or no effect except that in the last one-half hour there was a rapid rise in acidity to 76 at a period when the control showed an acidity of 32. A similar experiment on another patient gave practically the same results. Doses by mouth sufficient to give signs of belladonna poisoning (dry mouth, dilated pupil, etc.) raised the average acidity from 35 to 51.1. In all cases the motility was unaffected.

(2) *In cases with continuous secretion*.—In the control test a highly acid gastric juice persisted to the end of the experiment, 6¾ hours, though all the food had left the stomach at 2¾ hours. After the food had disappeared the acidity was higher than before, reaching 118 at 4½ hours. The average total acidity was 89.

The next day with the same patient, all food having left the stomach at 2¼ hours, one mg. of atropine sulphate was given at this time by hypo, and at one hour later the secretion had ceased. In the same patient fully atropinized by mouth for three days, the stomach was free from food at 3 hours, and the secretion ceased at 3½ hours. The average total acidity was 60 as compared with an average of 89 in the control, but in this case the averaging of acidities does not make a valid comparison, because in the control the highest acidity was reached during the period of continued secretion, *i. e.*, after digestion was finished, a period in which there was no secretion at all after atropine. In this case exploratory laparotomy had shown no lesion.

In a second case the secretion was still continuous at 5¾ hours, when the experiment was stopped. The average total acidity had risen from 85.3 to 94, the motility being unaffected. Then tincture of belladonna, 1 c.c. four times a day, was given by mouth for three days, when poisonous symptoms appeared. In a test at this time the secretion ceased at three hours, but the average acidity increased from 85.3 to 104.5.

We find then failure of atropine to lessen digestive secretion; and in continuous secretion cases, a failure to act except after the digestive period. Then it had an effect only if given by hypo-

dermic in maximum doses or if previously given by mouth to the stage of poisoning. It actually increased the acidity of the digestive secretion, and showed an inhibitory effect only on the abnormal continued post-digestive secretion.

In regard to the psychic secretion I have only the report of Rehfuß. In the early morning each of a number of men with a fractional tube in his mouth was set in front of a beefsteak and compelled to cook it though not allowed to eat it. The stomach secretion, withdrawn at intervals, ceased at 60 to 80 minutes, and ran as high a total as 240 c.c., with average acidity of 97.2. In large doses by hypodermic atropine cut down the acidity and the amount of secretion, but never caused complete disappearance of the secretion. It is possible that the drying effect of atropine in nose, mouth and throat, and the sensory effect of blunting the sense of smell, may have been important in the result.

(2) *Motor Functions*.—From a therapeutic point of view the only desired action of atropine on the motor functions is to overcome tetanic spasm at the cardia, at the pylorus, and at the site of an hour-glass contraction.

Working with strips from the antrum, pre-antrum, body and fundus of the stomachs of rabbits, cats and dogs, Smith found that solutions of atropine sulphate 1 in 1,000,000 and 1 in 100,000 invariably produced relaxation, whether the strips were from the longitudinal, the circular or the oblique muscles. He obtained the same effects on the pyloric and cardiac sphincters. Zunz and Tysebaert, working on the stomachs of dogs one-half hour after hypodermics of from 0.005 to 1 mg. per kilo, found the contractions weak and diminishing in strength, the effect in some cases persisting 5 to 6 hours. After 0.001 mg. per kilo the movements were normal though the tone rapidly fell. Auer and Meltzer have demonstrated that these effects are due to paralysis of the vagus terminals at Auerbach's plexus.

I have not worked with stomach strips, but in experiments done with Dr. C. C. Lieb using strips of the longitudinal muscle of the small intestine of dogs, atropine in large amounts completely abolished the tetanic contraction or cramps brought on by physostigmine and restored the peristaltic waves, but not the normal tone waves. From the work of others it is established that the same action takes place in the stomach. Ginsburg and

Tumpowsky, for example, found that hypodermics of 1/80 to 1/40 grain invariably in five to ten minutes inhibited the tetanic contraction produced by pilocarpine and physostigmine in the stomach, and restored the normal peristalsis. The inhibition was sudden and decisive and persisted for hours. The same results were obtained in the isolated stomach.

This conquering of tetanic spasm by atropine in doses which permit the vagus and splanchnic nerves to continue their ordinary influence on peristalsis, has led Cushny to surmise that these abnormal contractions, such as are seen in pylorospasm and colic, arise from some mechanism distinct from that which presides over ordinary peristalsis. This action on abnormal tetanic contractions is indeed *the only possible motor effect of therapeutic amounts of atropine*. It seems, therefore, essential to distinguish the two known motor effects, viz., that of abolishing abnormal spasmodic contractions, and that of abolishing the tone of the whole stomach wall. Only the former is possible or desirable therapeutically, and in all probability it is not accomplished by paralysis of the vagus endings.

Clinically and in Roentgen ray work, atropine has been much employed to overcome abnormal tetanic contractions. On cardio-spasm, which it is agreed is not a true spasm, the drug has little or no effect. In roentgenology it is quite generally employed to overcome pylorospasm and the spasm of hour-glass contraction. From fluoroscopic observations, A. E. Barclay reported that after the dose of atropine the spasm often let up quite suddenly, but in some cases the drug was without effect. From a number of the roentgenologists I have learned that quite frequently even large doses are without effect on pylorospasm. These men do not use small doses, but are accustomed to the employment of 1 to 1.2 mg. (1/65 to 1/50 grain) of the drug, and that hypodermatically; or some of them attempt to atropinize by 2 or 3 days' dosage. Within a few weeks I have had a case operated upon for cholelithiasis in whom the roentgenologist, finding an hour-glass stomach that did not change after a hypodermic of 1 mg. of atropine sulphate, insisted on a diagnosis of ulcer with cicatricial contraction. But at the operation there was no sign of either hour-glass or ulcer.

In Czerny's pediatric clinic, Ochsenius found that in infants enormous doses were necessary to overcome pylorospasm. For

instance, in one child a month old, to permit proper feeding he had to keep up intermittently for ten weeks an atropine dosage of 0.15 mg. (1/435 grain) five or six times a day, and in another three weeks old had to give the same dose eight times a day for a whole week. There is much evidence of the ineffectiveness in pylorospasm of any but large doses at any age, and in adults of the ineffectiveness of any but hypodermic doses; even then the relaxation is frequently not obtained.

Summary.—I.—*Acidity and Secretion.*—(1) In the ordinary hyperacidity case with cessation of secretion at the usual time, atropine or belladonna in maximum doses, either by mouth or hypodermic, has no useful effect on acidity or secretion.

(2) In cases with continuous secretion, a maximum dose by hypodermic half an hour before the meal did not lessen the acidity or secretion of the digestive period, but resulted in a stoppage of the secretion in a reasonable time after the food had left the stomach. A similar maximum dose had no effect.

(3) In cases with continuous secretion, repeated maximum doses of the tincture of belladonna by mouth for three days caused a pronounced increase in acidity during the digestive period, but a cessation of the secretion after the food had left the stomach.

(4) The psychic secretion is lessened, an effect not sought in therapeutics.

(5) The natural secretion of mucus is not affected.

We find then a complete failure of atropine to affect hyperacidity favorably, and a failure to diminish secretion except in continuous secretion cases. In these it does not depress and may even increase acidity and secretion during the digestive period; and it checks the continuous secretion only when given by hypodermic in maximum doses or when previously given by mouth to the stage of poisoning.

II. *The Motor Functions.*—(1) Atropine can exert two kinds of motor effects on the stomach: one, the abolition of tone in the whole stomach wall including the orificial sphincters, by action on the vagus myoneural junctions; the other, the abolition of abnormal spasmodic contractions as in pylorospasm, this effect probably not being dependent upon any action on the vagus terminals. (2) The latter effect is the only desirable one in therapeutics. It is a possible effect in some of the cases only, and

then only from maximum doses. (3) So far as I know the action of atropine on hunger contractions has not been studied.

Conclusions.—(1) In hyperacidity cases atropine has no useful effects in any dosage.

(2) In continuous hypersecretion cases it may check the secretion after the digestive period, but it does this in maximum doses only.

(3) In pylorospasm it may be useful, but in maximum doses only.

(4) In the doses usually employed it is wholly without effect on the stomach.

Pepsin.—By the U. S. Pharmacopoeia test with 1 in 3,000 hydrochloric acid at 125.6° F. (52° C.) pepsin is required to digest 3,000 times its own weight of coagulated egg-albumin in 2½ hours, *i. e.*, one grain of pepsin can digest at least 6¼ ounces. Prof. Gies of Columbia tells me that a specimen has been prepared two hundred times as strong as this, *i. e.*, one grain can digest 600,000 times its weight. What a wonderful substance to have so little use in medicine! It is inactivated by hydrochloric acid above 0.5 per cent. strength (U. S. P.), 0.7 to 0.9 per cent. (Hamburger and Halpern), and by sodium chloride solution of 2.5 per cent. strength. It is not only inactivated but is destroyed by alkalis, for example, di-sodium phosphate in one-half per cent. strength (Hamburger and Halpern), sodium hydroxide in 0.01 per cent. strength (Sollmann), and sodium bicarbonate and carbonate, magnesium carbonate and lime water (Hamburger and Halpern) when in sufficient amounts to make a persistent alkaline reaction. In the light of this destruction, one wonders how Abderhalden and Meyer were able to find active pepsin in all parts of the small intestine, and to suggest that this pepsin would be active in the digestion of protein wherever the intestinal contents should become acid.

In the stomach contents, Wiltrup found it absent or below normal in everyone of a thousand cases of achylia gastrica. Hernando and Alday found it absent in 3 and present in only very small amounts in 16 out of 22 cases of gastric cancer, and in normal or above normal amounts in 65 cases of hyperchlorhydria, 37 cases of gastric or duodenal ulcer and 85 cases of cicatricial stenosis of the pylorus.

It is evident then that the only possible cases for the use of

pepsin in therapeutics would be those of subacidity and achylia, whether cancerous or not. Its need is doubtful; but since pepsin digests protein only when this has been changed to acid albumin, if used at all it should be accompanied by a sufficient quantity of hydrochloric acid.

Pepsin preparations regularly have a milk-coagulating power. Whether this is due to admixture of rennin, or because pepsin and rennin are one and the same enzyme is a still unsettled question among physiological chemists.

Rennin is not a digestant, but it has the power to coagulate from 5,000 to 166,000 times its weight of milk in from one to several minutes. The rennin curd uses up 13 per cent. more calcium phosphate than the curd from hydrochloric acid (Harris) and is less dense and more readily acted upon by pepsin.

The function of rennin in the gastric juice is, therefore, to retard milk in the stomach by changing it to a solid, and to favor the digestion of its coagulable protein.

But in a medium strongly acid or more than slightly alkaline the rennin will not act. Therefore, on the one hand, in hyperacidity cases the curd formed is regularly the dense and comparatively indigestible acid curd and not that of rennin; and on the other, the addition to milk of more than a very little lime water or sodium bicarbonate, or as little as two grains of sodium citrate to each ounce, will prevent the rennin coagulation, and will keep the milk in its liquid and less digestible form. In highly acid stomachs, however, it may take considerable alkali to prevent the undesirable acid coagulation of milk.

If we add rennin to milk just at the time of swallowing, may we not find this a useful remedy, (a) in achylia cases with diarrhea, to coagulate the milk and so prevent its too rapid passage into the intestines, and (b) in hyperacidity cases to forestall the undesired acid coagulation?

Hydrochloric Acid.—The known functions of this acid in the normal animal stomach are: (1) To favor protein digestion and the disintegration of connective tissue. (2) To induce closure of the cardia. (3) To establish a normal intermittence of opening and closure of the pylorus. (4) To serve as anti-septic. (5) To aid inversion of the disaccharides. (6) To form secretions to stimulate the production of pancreatic juice and bile.

That the absence of hydrochloric acid is quite compatible with fair health and the maintenance of nutrition is proven by the frequency with which the existence of achylia gastrica is discovered after only insignificant symptoms or no symptoms at all. I found achylia present in one girl of 17 with complete lack of gastric symptoms, having tested her stomach merely because her grandmother and mother had shown achylia. For hydrochloric acid as a remedy only one use can be suggested, viz., to replace a deficiency of acid in the gastric juice. Whether introduced acid can so serve is our question.

(1) *Protein Digestion*.—It is an established fact that for pepsin digestion of protein acid is necessary. Therefore, in an achylia case, if we are to have any digestion of protein in the stomach we must supply hydrochloric acid as well as pepsin. Experiments done without pepsin regularly show a fair formation of acid albumin, and thus give hope that the addition of pepsin may ensure at least some degree of gastric protein digestion.

Crohn, in fractional experiments on man in achylia cases, both simple and those of pernicious anemia, found that it was not possible to have a sustained acidity from mouth doses unless these were frequently repeated during the digestive period. To mention some of his experiments, in an *emptied fasting stomach* he placed 40 minims of diluted hydrochloric acid mixed with 100 c.c. of water. The immediate acidity was: free 32, total 40. Successive specimens withdrawn at five-minute intervals showed rapidly diminishing acidity until at 25 minutes the titer was the same as before the acid had been given. When he administered 30 minims of diluted hydrochloric acid with an oatmeal test breakfast, the free and total acidities at 15 minutes were 8 and 20, and at 30 minutes 12 and 18, but at 45 minutes and thereafter had returned to 0 and 10 or thereabouts, the same as in the control experiment.

Then he administered repeated instead of single doses of the acid. Ten minims of the diluted acid every one-half hour during digestion gave a mild but definite rise in acidity which was sustained for $1\frac{3}{4}$ hours; and 10 minims every quarter-hour raised the average total acidity from 20 to 55. The emptying time was unchanged.

Leo, quoted by Crohn, gave achylia cases large doses of hydro-

chloric acid, equivalent to from 75 to 225 minims of U. S. P. diluted hydrochloric acid, and obtained increases in total acidity but rarely any free acid.

Spencer, Meyer, Rehfuess and Hawk introduced strongly acid solutions (0.542 and 0.4 per cent.) and found that the gastric contents had returned to normal at the end of one hour. This they attributed to a rapid emptying of the acid and a progressive neutralization of the excess of acid.

Rehfuess found that 10 or 15 minims of hydrochloric acid, properly diluted, made no detectable change in the gastric chemistry. However, with a constant supply of acid for two hours by a Murphy drip at the rate of 200 c.c. of 0.25 per cent. acid per hour, equivalent to 5 c.c. of diluted hydrochloric acid per hour, the curve of secretion rose to 33 per cent. of normal, though there was at no time any free acid.

All these findings indicate that if we can only give enough hydrochloric acid by mouth, we can at least hope to change our albumin to acid albumin and thus prepare it for pepsin digestion. But acid alone is practically useless for the purpose of digestion, and to judge of its merits as a remedy it must have all the factors for its action. In other words, we must administer pepsin with it. Moreover, the products of the action of pepsin and hydrochloric acid on protein are themselves capable of exerting a strongly stimulating effect on secretion.

Sippy makes a calculation that for the adult allowance of 100 gm. of protein a day, it would require 700 minims of diluted hydrochloric acid for full gastric digestion, and that on account of the sensitiveness of the mouth and throat to acid it would be impossible to swallow this amount. Of course, there is never, even in normal cases, full protein digestion in the stomach. Moreover, some proteins require less acid than others for digestion, *e. g.*, Hawk states that the best strength for the digestion of fibrin is 0.08 to 0.1 per cent., while the best for coagulated egg-white is 0.25 per cent. Joubert found in achylia cases that on giving hydrochloric acid at the same time as raw meat, connective tissue appeared in the stools. N. W. Jones was rather disposed to think that connective tissue in the stools was associated with a delayed stomach emptying time. From strengths of 30 to 40 minims in 100 c.c. of water Crohn noted the development of considerable mucus, presumably from some local irritant action.

(2) *The Emptying Time of the Stomach.*—That in achylia gastrica it is not infrequent to have a very rapid emptying time is well known. In 11 cases of pernicious anemia, C. P. Horner found that 6 had emptied at one hour, and in only one was food found at the two-hour period. N. W. Jones in a study of achylia, concluded that in a broadly built non-ptotic person achylia is usually associated with a too rapidly emptying stomach and in this class diarrhea is prone to occur; whereas in ptotic types the ptosis and atony prolong the emptying time even up to 7 hours or more, and in this class constipation is the rule. Goiffon states that 1.5 to 3 gm. of hydrochloric acid a day may act to cause pyloric closure and so retard the food in the stomach. Jones' experience with the use of acid tallies with that of the author. He says "many are symptomatically relieved by its use, while others experience increased sourness and stomach irritation."

There is considerable clinical evidence that in a fair number of achylia diarrheas, hydrochloric acid is a successful remedy, and may not this be due to the restoration of the normal pyloric closure through the acid reflex, and the consequent retardation of the food in the stomach? This, it is to be remembered, is a desired effect only in those cases with rapid emptying and diarrhea, and it is to be avoided if possible in those cases with flatulence or constipation. Crohn found the emptying time normal after doses of acid, but he does not state that any of his cases were of the diarrheal type.

It has been stated that in some achylia cases the diarrhea may be checked by the administration of pancreatin. May not part of the value of hydrochloric acid lie in its power to enhance the pancreatic secretion, through the formation of secretin, and thus to ensure more thorough digestion of proteins in the intestines? For, though achylia gastrica is not ordinarily accompanied by pancreatic achylia, yet for certainty of digestion the rapidly passed food may require abnormal amounts of the pancreatic ferments.

(3) *The Antiseptic Action.*—In an achylia case it is quite common to find pronounced intestinal putrefaction. In both diarrhea and constipation cases this may be attributed, in part at least, to the failure of the achlorhydric contents to arrest the development of gas-forming organisms and to destroy the sundry proteolytic and pathogenic germs, thus permitting their passage

into the upper intestine. In diarrhea cases intestinal putrefaction might also be accounted for by the fact that the ferments have too short a time in which to break down the protein before it reaches the usual region of abundant germ life in the intestine, and so the protein furnishes pabulum for the bacteria of the colon.

(4) *The Effect on Pancreatic Secretion and Bile.*—It is a well-established fact that, in the normal case, the hydrochloric acid of the stomach is an important factor in the production of secretin which stimulates the secretion of pancreatic juice and bile. What takes the place of hydrochloric acid in the achylia case, which does not lack pancreatic ferment, has not been determined; but if free hydrochloric acid or combined acid, which is really a loosely combined protein salt, can be passed into the duodenum, even for a few moments as the result of introduced acid, may not this result in the formation of ample intestine secretin, and so provide the normal stimulus for the production of pancreatic juice and bile?

Summary.—In achylia cases, from experiments based on the introduction of hydrochloric acid alone, without its natural congener pepsin, it is evident that with single doses of swallowable strength the acid titer of the stomach contents may be distinctly raised. The results that might be expected from this are: (1) successful protein digestion in the stomach when sufficient pepsin is also introduced, (2) slowing of the emptying time of the stomach by re-establishment of the pyloric closure reflex, (3) restoration of the normal antiseptic action in the stomach, (4) the proper formation of secretins.

Possible drawbacks to its use are: The establishment of pyloric closure in an already slowly emptying stomach, the production of stomach irritation, and the development of a mineral acidosis from its daily use over long periods of time. That it can produce *acidosis* has been demonstrated in dogs by a number of observers, and recently in man by Marriott and Howland. They gave 500 c.c. of decinormal hydrochloric acid in one day to each of four normal men who ate their usual diet. This amount would represent 273 minims of diluted hydrochloric acid, a very large amount. On that day there was a distinct increase in the ammonia coefficient in the urine and at the same time an increase in the titratable acid in about the same proportion. Thus in a single day from swallowable amounts in man ammonia was de-

flected from the usual course of nitrogenous metabolism, and a certain degree of acidosis was produced.

Stehle administered hydrochloric acid to dogs by mouth, and found an increased excretion of sodium, potassium, calcium, and magnesium. In the case of sodium and potassium, however, a subsequent compensatory retention took place. It has been figured that among the other effects there is a loss of calcium from the bones.

It is of interest that Ginsburg, Tumpowsky and Hamburger found hydrochloric acid in 0.5 per cent. concentration to be without effect on the hunger contractions.

Conclusions.—(1) In cases of achylia gastrica, whether or not accompanying pernicious anemia, a deficiency of acid may be partially overcome by hydrochloric acid medication.

(2) For digestive purposes hydrochloric acid should always be accompanied by pepsin.

(3) In the achylia with diarrhea acid promises a more noticeable result than in the achylia without diarrhea.

(4) When acid produces sourness and stomach irritation its use should not be continued.

(5) To avoid acidosis alkalies should be given during the same period, though not at the same time as the acid, the amount required being judged by the effect on the urine reaction.

To avoid the trouble of frequent medication in cases of achylia gastrica I have found it of value to have the patient take at the main meal most of the protein of the day, or at least of the more putrefactive proteins such as eggs and flesh foods, and to give the acid and pepsin with this meal only. For practical reasons liquid medicine cannot be taken for any length of time after the meal, but doses of 20 or 30 minims of diluted hydrochloric acid and a few grains of pepsin in a full glass of water may be taken with the meal and frequently at half and even one-hour later.

The Solid Hydrochloric Acid Preparations.—In solid form there are marketed certain drugs purporting to contain hydrochloric acid available for digestive purposes. The best known are *oxyntin*, a protein compound of hydrochloric acid, and *acidol*, which is chemically betaine hydrochloride. In a careful research under the auspices of the American Medical Association, Long found that acidol dissociates in an aqueous medium and supplies hydrochloric acid; but that oxyntin holds scarcely enough acid

for the digestion of its own protein and cannot therefore supply any for other digestion.

Nitrohydrochloric Acid has frequently been employed by the older physicians. It is made by mixing hydrochloric and nitric acids, a violent reaction taking place, and the acids being split up to form nitrosyl chlorides and chlorine. There is a slight excess of hydrochloric acid, so that nitrohydrochloric acid of the Pharmacopoeia is a liquid containing free hydrochloric acid, free chlorine, and nitrosyl chlorides. It hardly seems worthy of a place in the *Materia Medica*.

Bitters.—In Carlson's tests on a young man in good health, who had an esophageal obstruction and a permanent gastric fistula for feeding purposes, bitters were administered either by stomach 15 to 30 minutes before meals, or by mouth 10 minutes before the meal. The subject was in the habit of taking his food by mouth, chewing it and then placing it in the stomach through the fistula. The bitters used were the tinctures of gentian, quassia, calumba, humulus and condurango, and the elixir of iron, quinine and strychnine. The tests numbered 50 with bitters taken into the mouth, 35 with bitters placed in the stomach, and 50 without bitters for control.

When the bitters were taken by mouth they could not be retained long because of the salivation induced, and had to be expectorated. Their effect on the appetite of this young man in good health was too slight to be of moment, though it was noted that at the evening boarding house meal, which was never relished, the effect of the bitters was to make an already undesired meal still more undesirable.

When the bitters were placed directly in the stomach, small amounts were found to have no influence on the quantity of the psychic secretion or on the acidity or pepsin concentration of the gastric juice. When large amounts were used, Carlson's results agreed with those of other observers that their action in the stomach itself was rather to retard than to increase the activity of gastric digestion.

With tests on healthy dogs Carlson further came to the conclusion that bitters acting either in the mouth or in the stomach have no effect on the secretion of gastric juice.

In another series of experiments Carlson and his colleagues showed that bitters acting in the stomach alone have no appre-

cial influence on the hunger mechanism as distinguished from appetite, and that when taken by mouth in the usual way they inhibit gastric tonus and hunger contractions in direct proportion to the intensity and duration of stimulation of the nerve-endings in the mouth. In other words, *in normal people*, so far as they influence the hunger mechanism directly, the bitters cause inhibition or depression of hunger.

Arguing from these experiments Carlson takes the ground that the whole value of bitters in medicine is mental and that they are in the same class as any inert but widely advertised patent medicine. He thinks that their continued use depends on two beliefs of the patient, namely, that they will promote appetite, and that anything with a strong and bad taste is strong medicine and therefore good medicine.

On the other hand, Hoppe noted that in a sick dog the use of a bitter was followed by an increase in the quantity and acidity of the gastric juice; and Moorhead found that while in normal dogs bitters had no influence on appetite and no influence on secretion or a depression of it, nevertheless in dogs made cachectic by daily bleedings to produce a chronic anemia, they caused a distinct and significant increase in both appetite and secretion. Placed in the stomach without touching the mouth they had no appreciable influence. Barisoff gave tincture of gentian to a dog with the end of the severed esophagus opening outside so that substances swallowed did not reach the stomach. He followed this with a meal, and in 6 tests without the bitter and 6 tests with, found that after the bitter the average amount of gastric juice increased 30 per cent. If he gave the bitter as long as 20 minutes before the meal this effect was not obtained. An excess of bitter checked the secretion. I myself have noted in many cases of achylia gastrica that bitters have the power to create or increase appetite, though without effect on the gastric secretion.

From these experiments and my own personal experiences I would register my opinion that in many persons with subnormal nutrition, especially in those recovering from an acute illness, bitters have a real value in promoting appetite, and that this action is not dependent either on the patient's belief in the efficacy of the drug, or on the drug's power to affect stomach secretion.

Conclusions.—A bitter is useful as an appetizer for those with

subnormal nutrition, as in convalescence from acute illness, provided that it is taken not more than five or ten minutes before the time for eating. It acts in achylia gastrica as well as in cases with gastric secretion. It should be administered in just sufficient dose to give a strong bitter taste, and not in large enough amounts to have a depressent action in the stomach. If the patient is in a state of normal nutrition but psychically disturbed about eating it will be useless. If the appetite is already normal, the bitter may not only fail to increase appetite but may even lessen it. If the stomach and bowels are deranged a bitter may nauseate. The effect on appetite is solely the local one on the taste buds, therefore it cannot be obtained if the bitter is hidden in capsules or coated pills.

Cerium.—The oxalate, which is the one salt employed, was studied by Baehr and Wessler. They found that it is non-poisonous to dogs even in doses of 50 grams, and that its action is mechanical as a protective to the gastric mucous membrane. Administered in advance it would check the vomiting from a local stomach irritant, such as ipecac, but had no influence on the vomiting from a central emetic, such as apomorphine. They state that the drug is useless in the small doses usually administered, and recommend that it be given in doses at least as large as those of the bismuth salts.

Bismuth.—The bismuth salts in common use are the subcarbonate, the subnitrate and the subgallate. The subnitrate is crystalline, and because of this fact probably less bland than the others, which are amorphous. Contrary to the general belief they are all without astringency, that is to say they do not cause shrinkage of the tissues with which they come in contact.

Being basic salts they have the power to take up acid. Theoretically, of *bismuth subnitrate*, one gram will neutralize 2 c.c. of diluted hydrochloric acid, or the acid of at least 40 c.c. of gastric juice, with the formation of bismuth nitrohydrochloride. But the change takes place very slowly, and Böckmann has demonstrated that in such acid concentrations as are found in the stomach the subnitrate possesses but little acid neutralizing power. Of *bismuth subcarbonate*, one gram will neutralize 4 c.c. of diluted hydrochloric acid or the acid of at least 80 c.c. of gastric juice, with the formation of bismuth chloride. It does not change to bismuth oxychloride as so frequently stated, for this basic

salt cannot form in an acid medium. Bismuth subcarbonate changes somewhat more rapidly than the subnitrate, but not rapidly enough to justify its use as an antacid.

In a study of test meals with the addition of bismuth subcarbonate, and fractionally extracted every fifteen minutes, Crohn reported that 2-gram doses given directly after the meal caused a diminution in acidity without any compensatory increase (as found after alkalis) in the acid secreted. According to a published chart he administered the salt half an hour after the meal, and up to the $1\frac{1}{2}$ -hour period found the acidity slightly higher than in the control. But at $1\frac{3}{4}$ hours he got an acidity of 40 as compared with 70 for the control, and at $2\frac{1}{4}$ hours 50 as compared with 68. There was a slight retardation in the emptying time. In a duplicate experiment with a slightly greater dose the acidity was depressed from 56.8 to 45.2 without delay in emptying or any evidence of the secondary rise in acidity which regularly follows the administration during the digestive period of the alkalis and alkaline earths.

Some years ago in a number of instances I administered doses of 2 grams of bismuth subnitrate just before or just after the test breakfast, and though at the end of the hour the stomach contents usually showed a lessened acidity, and also a lessened secretion as determined by the Matthieu-Rémond method, there were a few cases in which the acidity was unchanged. Unfortunately the protocols and the case records of these experiments were destroyed, so that I am not able to figure a reason why the bismuth should fail in these instances. In every case it was noticed that at the end of the test breakfast hour the bismuth was uniformly mixed with the extracted stomach contents, and that it had changed from a heavy powder to a flocculent substance that settled slowly with the food. On two occasions I administered to dogs with their food doses of 2 grams and 8 grams respectively of bismuth subnitrate colored red with carmine, and on removing the stomach and intestines three and seven hours later found the colored bismuth in this same flocculent and comparatively light state, partly mixed with the food residues, but mostly coating very uniformly the whole mucous membrane. In the dog killed at three hours it coated the stomach and small intestines throughout, except the first three or four inches of the duodenum. In the dog killed at seven hours it coated the

whole jejunum and ileum. The coating stopped short at the ileocecal valve and there was no macroscopic evidence of bismuth in the colon.

Crohn's results with bismuth subcarbonate and mine with bismuth subnitrate would seem to be of the same nature, and would suggest that the action of either salt is not antacid but rather protective by coating the mucous membrane. Their power to spread over a large surface of membrane is almost phenomenal. Crohn's work establishes the fact that while the bismuth salts affect the secretions they do not essentially change the motility. By their protective action these bismuth salts would seem to have a value quite equal to that of cerium oxalate in preventing vomiting from local stomach irritants, such as ipecac.

Toxicology.—By mouth the bismuth salts, even in large therapeutic doses, are not ordinarily poisonous, though Kohn reported a case of stomatitis and other manifestations of metallic poisoning from doses of 0.3 gm. (5 grains) given four times a day. But poisoning from the use of a bismuth paste in the treatment of sinuses is not uncommon. One fatality resulted from less than 10 grams of bismuth subnitrate mixed with vaseline. Therefore it may be conceived as possible that if the bismuth salt given by mouth should be retained on a raw area such as an ulcer, metallic poisoning may occur. As demonstrated by the Roentgen rays such retention is unusual, but last year Dr. Kaufmann told us of a case of hematemesis to which Naunyn had given 25 grams of bismuth subnitrate, and in which at the post mortem von Recklinghausen took out of the crater of the ulcer 20 of the 25 grams of bismuth.

The symptoms have the characteristics of poisoning by the heavy metals, viz., stomatitis, salivation, a violet, blue-gray or blackish line on the gums, nausea, vomiting, diarrhea, and prostration.

After bismuth subnitrate, but no other bismuth salt, another form of poisoning has occurred, namely, nitrite poisoning, this being due to the formation of nitrous acid. Most of these cases have resulted from the ingestion of large amounts of bismuth subnitrate for Roentgen ray work; but there are a few that have occurred from the medicinal use of the salt. Böhme, for example, reported that after giving an 18 months marasmic infant several grams by mouth and two days later a similar dose by

rectum, the child three hours after the last dose suddenly developed abdominal pain, diarrhea, cyanosis and dyspnea, and died in half an hour. The blood and pericardial fluid gave tests for nitrous acid, and the blood for methemoglobin. Böhme found that when he mixed bismuth subnitrate with feces, nitrous acid was formed, and that when he placed this mixture in a rabbit's intestine, the urine showed nitrites. In one fatal case E. Meyer demonstrated nitrites in the urine, blood and pericardial fluid.

Silver Nitrate.—Before leaving the subject of drugs I wish to put on record a case of argyria that I have just found on my service, with a universal metallic slaty look to the skin, which came on after taking only $\frac{1}{4}$ grain of silver nitrate three times a day for two months.

I also this year had an autopsy on a similar case of argyria, in which the viscera were much discolored with silver deposits. This resulted from the application only once daily of a caustic silver stick to the mouth of a fistula. The period of application I do not know. This may point a moral for men who use silver nitrate in gastro-enterology.

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DISCUSSIONS.

DR. REHFUSS: The first question I would like to remark upon is the last one of the paper. I do not quite agree with Dr. Bastedo regarding the effect of atropine on the stomach. It is true that if you will test the total gastric curve and the total amount of gastric work, atropine apparently has no effect. However, in our work on estimating the different portions of the curve, namely the psychic and the chemical portions of the curve, and performing the experiments on the psychic secretion in which there were no chemical stimuli in the stomach whatsoever, we were able to demonstrate that atropine does reduce that secretion, and it has reduced the secretion approximately two-thirds and the acidity more than one-half. Now, that is on the psychic secretion. We have given atropine by mouth and hypodermically. I have given it in doses as high as 1/50 of a grain and studied the effect; and the sum total of the effect is certainly less in the majority of instances on the entire gastric secretion. I believe, as Dr. Bastedo's paper states, that atropine is to be used as a muscle drug rather than a secretory drug. Regarding the question of the action of acid on the stomach, I feel about it as Dr. Bastedo does. We found practically no effect whatsoever. We found, however, that if you introduce a large quantity of acid into the stomach—in the

highly acid stomach—like one-half of one per cent. of hydrochloric acid, there is ensuing reduction to what I call the equilibrium point.

Bitters.—We practically all feel the same about it. The stimulating effect of bitters has no effect whatever on the stomach.

DR. CROHN: I am a sort of pessimist on the subject of the therapy of gastric secretion. The stomach seems to come back to an equilibrium curve which it seems very hard to budge or to alter. With the experiments with alkalis, it has been shown that you can repeatedly treat your cases with alkalis, following them from day to day for many weeks. The giving of alkalis or acids in repeated doses—in small doses—is hardly practical. It is very difficult to carry out in your practice. It can be done, and we have done it at the hospital. We have done it repeatedly, giving the patient a great big pitcher of dilute alkali and bicarbonate of soda and asking them to sip it from time to time, and you do get a certain amount of neutralization of acid. There were many cases of achylia gastrica which I have experimented on, giving hydrochloric acid in repeated and divided doses. There were no diarrhea cases in those cases at all. The clinical improvement in hydrochloric acid cases is so marked that one cannot miss it.

I am not one of those who believes that there is any pancreatic achylia or any defect of pancreatic secretion in cases of achylia gastrica. The hydrochloric acid may be completely absent in the stomach, and the ferments may be and are usually completely absent in the stomach, and yet nobody will have any difficulty in convincing himself that the pancreatic ferments are all three present, and present in full strength.

DR. STOCKTON: In reference to the action of cerium and bismuth, a distinguished dermatologist some years ago called my attention to the effect of certain zinc salts topically applied in cutaneous diseases. For instance, zinc carbonate in an ointment will act favorably in one case, while zinc oxide will act unfavorably. In another case, where the zinc oxide will act well, the zinc carbonate will act badly. I have come to the same conclusion in regard to the use of cerium and the salts of bismuth, of kaolin, of charcoal, of chalk, or other substances used as merely local applications to the gastric mucosa. There is no doubt, from my experience, that one person is favorably affected by cerium where another patient, who is apparently suffering from the same trouble, is more favorably acted upon by bismuth. I have not been able to convince myself that there is any distinction to be made between the action of bismuth subnitrate and bismuth subcarbonate. But it does seem that the effect of bismuth subgallate differs from that of bismuth subcarbonate. I think they are entirely local in their effects, and according to the same reasoning, just as zinc salts vary in their action upon the skin in different individuals having the same affection, so bismuth, cerium, etc., differ in their effects upon the mucosa, according to the individual.

DR. BASTEDO, closing: I am very glad that the discussion was brought out, because it is worth as much as the paper. I have nothing more to say. Some of the points mentioned I have discussed in the paper, which was curtailed because of lack of time. I agree with these gentlemen that the clinical findings have not always corresponded with the theoretical conclusions which might be drawn from laboratory experiments with these particular drugs, and yet sometimes the pendulum swings. The pendulum swung very strongly against hydrochloric acid, and then it has come back a little bit, and the pendulum swung very strongly in favor of atropine, and it is coming back a little bit. In treating a large number of cases of mucous-colitis I have not found any value whatever from atropine in quite large doses or small doses for long periods of time, except in so far as it affected the spasms in the bowel.

FOOD FACTORS IN GASTRO-ENTEROLOGY.

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If one were to interpret the present-day scope of gastro-enterology from the contents of the papers which were presented at the last annual meeting of this Association, it would, I suspect, be difficult to find a place therein for a topic such as I am venturing to discuss at this time. It seems to me, however, that a discipline which relates to "the normal and pathological conditions of the digestive organs" cannot fail to find some interest in the rôle of the food, for the transformation and transport of which these organs exist, or in the phenomena of nutrition, for which the function of digestion is an indispensable preparation.

There are growing manifestations of a more lively interest on the part of the so-called clinical worker in the contributions of the scientific investigators of the laboratory. It is a platitude to remark that no detail of the fundamental physiology and biochemistry of the alimentary processes should be deemed too insignificant to deserve consideration from the gastro-enterologist. The modern researches on the movements of the digestive canal, the secretions which reach it and the chemical changes which proceed within it are finding applications in diagnosis and therapy of gastro-intestinal disorders. Unfortunately, rational dietetics founded upon the newer knowledge of the chemistry of foods and nutrition has not yet received the discriminating study and advocacy on the part of practical clinicians that its importance unquestionably warrants. We may frankly admit that dietetics is at present far from being an exact science, so that dietotherapy is anything but an accurate art. Nevertheless, the limitations of our knowledge, based, in part, on empiricism and modified by that factor of uncertainty described as personal idiosyncrasy, need not disparage the recourse to novel therapeutic viewpoints and new procedures which new scientific facts are likely to initiate when they are given a sympathetic hearing.

The current attitude of aloofness from sane and helpful diet-

etics in medical practice is largely engendered by ignorance and indifference, conditions which the recent progress in this field should help to dispel. The widely divergent methods now in vogue in the treatment of gastric ulcer—methods which range from enforcement of complete physiological inactivity and absence of food in the upper alimentary tract, as the one extreme, to liberal feeding, intended to secure better healing through improved general nutrition, as the other extreme—illustrate the place which a fundamental understanding of food factors should have in rational gastro-enterology. Indeed, there are few, if any, specialized departments of medicine which would fail to benefit by greater attention to the fitness of the body as a whole. The physiological functions are so widely correlated and interdependent that there is oftentimes danger of overlooking the major advantage because of a concentration of attention upon a more obvious minor defect.

Food and Disease During the War.—The exigencies of the World War and the unusual food situations created by it have compelled thousands upon thousands of persons to alter their traditional modes of nutrition or their customary dietary habits. This change has not been instituted without many protests. Even the finest spirit of patriotism could not always repress misgivings regarding the physiological wisdom of the enforced or recommended innovations of diet. All physicians, probably, can substantiate my assertion that the medical profession received many anxious appeals to corroborate the alleged safety of the dietary restrictions which the governments demanded in the interest of the win-the-war policy. Many of the proposals cost the responsible officials of our Food Administration no little anxiety lest the substitutions and economies and the food sacrifices and modifications of diet might in any way alter the well-being of our population. During the war we have learned the possibility of using more than one cereal to advantage; we have succeeded in lowering the consumption of meat without apparent detriment; we have reduced the intake of sugar to the plane where it represented a condiment rather than a food; we have restored the conserved and less expensive vegetables to a worthy place in the day's food—these and other dietary changes have been instituted amid the fears of the devotees of custom in foods.

It must not be assumed, however, that the enforced war-time

restrictions have been attended everywhere with equal nutritional success. The effects of the difficulties in the food situation in the Central Empires of Europe upon the health of their peoples have recently been reported by a no less competent observer than Prof. F. Kraus,¹ of the Charité Hospital in Berlin. Addressing the members of the medical profession, he referred, in the domain of gastro-enterological disorders, to the numerous cases of intestinal dyspepsia provoked by the monotonous, voluminous diet preponderating in difficultly digestible carbohydrates; to the gastric dyspepsias attended with achylia, less often with diarrhea; to the intestinal fermentations; above all to the induced constipation, flatulence and the induced or augmented catarrhal condition of the bowel; and likewise to an increased incidence of ulcer. Furthermore, Kraus alleged that, among numerous persons in all walks of life, the monotony of the crude diet, the lack of gustatory stimuli and suitable food accessories, and possibly of vitamins, have induced a degree of anorexia or food satiety which has ultimately been followed by inanition. Surely the recital of these untoward experiences affords a cogent argument for a better appreciation of the specific rôle of the individual nutrients not merely in the physiology of digestion, but also in the clinic of gastro-enteric disease.

Some Newer Viewpoints.—If we turn to the innovations of viewpoint which the newer physiology offers as worthy of the attention of the gastro-enterologist the modern conception of secretion becomes conspicuous. Twenty years ago, when this Association was still in its infancy, the mechanism of the control of the glandular activities, which are so important in relation to alimentation, was believed to reside in the nervous system. Secretion was conceived to be initiated or inhibited, as the case might be, through the medium of nervous impulses. Today a humoral path of stimulation through the intermediation of chemical substances—hormones or succagogues carried in the blood stream—is becoming well recognized. I cannot but believe that if the various chemical stimulants to secretion, particularly as they occur in our diet ingredients, were better known and appreciated, the information would find more frequent application, particularly in relation to the promotion of gastric secretion. It is not too much to hope that selected foods and related substances may before long be expected to accomplish for secretion

some of the effects which drugs are at present often called upon to bring about.

Vitamines and Gastro-Enterology.—The word “vitamine,” scarcely five years old, has already become an expression to conjure with. The vague and mysterious characteristics with which it is at present endowed will, I feel confident, gradually acquire a more specific significance as the potencies of “the hitherto unidentified food factors indispensable to normal nutrition” are elucidated by further research. Consider the following striking experimental demonstrations: An animal is placed upon a diet consisting of isolated proteins, carbohydrates, fats and inorganic salts—the traditional mixture of nutrients which the physiology of our teachers has led us to expect to be adequate for the body’s needs. Nutritive failure and decline will inevitably ensue, attended by a variety of symptoms, perhaps including those seen in beriberi. An exceedingly small dose of brewers’ yeast or a chemical fraction prepared therefrom, or a small allowance of a vegetable like the tomato, spinach or carrot, or an addition of milk or of any of a large variety of naturally occurring foods to the dietary, will bring a restitution of health with a speed and completeness that is little short of marvellous. We are face to face in such instances with the nutrition-promoting potency of something which cannot be expressed in terms of the hitherto recognized nutrients. A ration compounded of washed skeletal muscle (beefsteak), carbohydrate, fat and salts leads to analogous nutritive failure; whereas, the substitution of liver or kidney tissue for the meat, in an otherwise unaltered diet, ensures uninterrupted well-being. In terms of the current hypothesis we have become accustomed to say that the yeast, the vegetables, the milk, liver, and kidney contain an essential food factor or vitamine.

Again, if in an otherwise adequate dietary the sole source of fat is represented by lard or one of the familiar vegetable oils, nutritive disaster will ensue sooner or later. During the period of pronounced malnutrition serious eye disease may arise as one of the intercurrent symptoms. The introduction of a small amount of milk fat, liver fat (as in cod-liver oil) or the oil of some of the vegetables, as Osborne and I have lately demonstrated, brings about an almost magical restitution of health. Here evidently we are concerned with the presence in certain of the fats

or fat-like mixtures of another potent property distinct from the vitamine already described. To these illustrations so striking that they are not easily forgotten by one who has witnessed the surprising remedial transformations induced by seemingly insignificant quantities of certain food products, may be added the presumably distinct antiscorbutic properties of certain natural foods.

The purpose of these fragmentary references to significant features in the recent studies of nutrition is not to emphasize primarily their broader dietary significance. I desire rather to offer the suggestion that dietary deficiencies, such as are exemplified by the lack of suitable vitamins in the ration, may affect the gastro-enteric tract quite as probably as other parts of the organism which are represented in the now recognized so-called deficiency diseases. The gastro-intestinal symptoms in pellagra afford a specific illustration. At the present stage of our knowledge the gastro-enterologist cannot afford to overlook the possible rôle of the vitamins in the functions of those parts of the organism with which he is most directly concerned.

In a recent interpretation of the nature of appetite Carlson² wrote:

"Appetite depends not only on the memory process of past experience, with palatable food as positive phases and the memory process of removal of hunger pangs by feeding, but the presence of this memory process in consciousness depends on certain conditions of the alimentary tract. When the stream of afferent impulses from the alimentary tract and possibly other visceral organs becomes altered in quantity or quality from the normal these impulses suppress or render impossible the existence of appetite. Hence it would appear that the existence of appetite is an indication of a proper condition of the alimentary tract to handle the food in the way of motility and secretion, and that this is the chief biological significance of appetite. The production of appetite gastric juice is of secondary importance and of practical significance only in cases of extreme impairment of gastric secretion."

We have frequently observed that one of the conspicuous manifestations of a dietary regimen deficient in certain types of vitamins is a diminished food intake. The feeding of vitamine-containing products almost always results in an improved appetite, if one may judge this by the resulting ingestion of increased

amounts of the same ration which was refused on the vitamine-free regimen. Precisely what the decisive relationships here are—whether improved appetite induced by the vitamine leads to better food intake and hence better nutrition or whether nutrition improved by the potent food factor results in better appetite—can scarcely be decided at the present time. At any rate the possible bearing of vitamins on the problem of appetite and alimentary well-being should not be overlooked by those who are interested in the physiology and pathology of the gastro-enteric tract.

Protein Factors—In considering the rôle of albuminous substances in nutrition the gastro-enterologist is confronted with the unlike digestibility of proteins from different sources. Thus the proteins of some of the legumes, notably beans, are admittedly more resistant to the enzymatic digestive changes than is the case with proteins from many other sources of both animal and vegetable origin in the alimentary canal. Why this is true remains to be elucidated. The tendency for native egg-white to be poorly utilized in the digestive tube has been emphasized by the investigations of Bateman³ in our laboratory. He has summarized the objections to the use of raw eggs, which is still widely advocated by some physicians, as follows:

“A substance which fails to stimulate a flow of gastric juice and is antipeptic, which hurries from the stomach, calls forth no flow of bile and strongly resists the action of trypsin, which is poorly utilized and may cause diarrhea, has evidently little to recommend it as a foodstuff of preference for the sound person, let alone for the invalid. And when the native protein needs only to be coagulated at 70° in order to obviate almost all the effects mentioned, there appears still less reason for using it uncooked. Other considerations strongly support this conclusion.”

Another illustration of the resistance to digestion on the part of a protein substance, presumably complete in its chemical make-up, is furnished by wheat bran. If further evidence were needed to demonstrate the failure of its protein to be utilized one could cite the latest experiments on man by Holmes.⁴ Even when wheat bran was ground very fine so as to be rendered more pervious to the digestive secretions the “coefficient of digestibility” did not exceed 45 per cent. Indeed, the advocacy and success

of wheat bran as a laxative is based in part at least on this resistance to alimentary digestion.

The three instances just cited exemplify the objections to the use, as nutrients, of protein products known to be difficultly digestive because of either chemical composition or physical texture. Such foods must often be excluded in dietotherapy when an undigested alimentary residue, susceptible to microbial changes, with the possible attendant symptoms of intestinal disorder, needs to be avoided. On the other hand, it must not be assumed that every protein which is digested with great readiness and completeness in the alimentary tract is on this account an ideal component of the ration. Gelatin is conspicuous for the readiness with which it yields to the proteolytic enzymes of the intestinal canal; despite this easy digestibility, however, it is an "incomplete" protein in the sense that it fails to furnish some of the amino-acids that are indispensable for perfect nutrition. This fact has long been known; and while the use of gelatin as a supplementary food can only be commended, its limitations as the sole source of nitrogen deserve to be reiterated at a time when a concerted effort is apparently being made to increase the consumption of the product.

Again, there are instances of proteins or protein mixtures which are not absolutely defective or incomplete in the sense referred to in the discussion of gelatin, yet are comparatively deficient in the yield of certain "building stones" essential for nutrition. Thus the proteins of wheat flour are easily digested and utilized in the usual sense. Compared with many other proteins, however, they are not economical sources of the nitrogenous nutrient units. Osborne and I⁵ have recently demonstrated that flour used as the sole source of protein is inferior to many other foods in maintaining adult rats, and especially in promoting the growth of the young. But when the proteins of wheat are fed in combination with about one-third of their weight of the proteins furnished by eggs, meat or milk these cereal proteins are so greatly enhanced in value that flour is thus used most advantageously. In fact, by far the greater part of the flour used in this country in every-day life is actually eaten in combination with those food products which successfully supplement the nutritive deficiencies of its proteins. Adequate nutrition means something more than calories and digestible nutrients; the com-

ponents of the diet must be appropriate in quality as well as quantity and all of the essential units must be represented.

The Inorganic Elements.—In a recent text-book it is stated:

“The mere enumeration of a few of the important uses of the inorganic elements brings out strikingly their significance. The multiplicity of their function has likewise rendered the study of these substances difficult, for with one element having a varied function its removal from the diet may be responsible for many secondary reactions which will mask the direct result.”⁶

Despite such generalizations it must be admitted that our knowledge of the functions of the inorganic elements in the body is still very fragmentary and indefinite. As Osborne and I have written elsewhere:⁷ “Although there is almost unanimity of opinion regarding the energy needs of the body under different circumstances of age and activity; although the current estimates of the minimum amount of protein required per day seem to be defined within reasonably narrow limits; although the functions of fat and carbohydrate and the possibilities of their interchange are beginning to be understood; there is no adequate experimental basis whatever to permit tenable statements regarding either the indispensability or the minimum requirement of any of the inorganic constituents of the dietary, with the possible exception of calcium and phosphorus. Statistics show enormous divergencies between the mineral intakes of people in different regions; but these appear to be the fortuitous results of widely unlike diets, including water (as is the comparative dissimilarity in the fat and carbohydrate content of the diets of peoples living respectively in a tropical or frigid climate—differences enforced by the unlike character of the available food supplies) rather than the expression of unlike metabolic needs. A beginning has hardly been made in this field of investigation.”

The need of certain elements for structural purposes in the body is obvious. The possible part which some may play in the obscure rôle of “maintaining osmotic equilibrium” is less certain, and when the assumed balance of acids and bases is discussed the border-line of accurate knowledge respecting the individual elements is reached. In studying growth under conditions in which the specific inorganic content of the ration could be fairly well controlled, each element being increased or diminished as desired, Osborne and I found that a lack of calcium or phos-

phorus promptly effects untoward results. The need of the other familiar inorganic elements, however, appeared to be unexpectedly small. This applied particularly to chlorin, and is presumably true of some of the other elements like sodium, potassium and magnesium. As we have pointed out: "That these may to some degree be essential to the adjustments of neutrality regulation is indicated by the failure to grow when both sodium and potassium were practically excluded from the diet, whereas growth was nearly or quite normal when only one of these elements was missing. That these elements take part in the processes regulating the neutrality of the body fluids is to be assumed from what has been learned by experimental work *in vitro* along these lines, and also from the fact that our experiments with diets essentially free from both sodium and potassium have led to nutritive failure." We concluded that in the long run much smaller quantities of those inorganic elements which can be husbanded will be required for well-being than of those which are needed for the maintenance of neutrality, and hence are continuously eliminated, wholly apart from any quantity necessary for the construction of special tissues like bone or for the production of milk.

The gastro-enterologist is peculiarly interested in the rôle of chlorin in the organism because of the indispensability of this element in the elaboration of the gastric juice. Common salt has frequently been prescribed to supplement diets presumably poor therein, with the avowed object of averting any failure of hydrochloric acid secretion, particularly because anacidity has been reported as a consequence of a salt-free diet. It must now be borne in mind, however, that when the intake of chloride is restricted the output promptly decreases. In starvation it is almost *nil*. Whatever chlorin is secreted into the stomach is subsequently reabsorbed and consequently conserved. The feces in health carry away little if any chloride. Referring to observations on man, Sherman states that even when there was complete deprivation of salt during ten to thirteen days the total loss did not exceed 10 to 15 per cent. of the amount estimated as usually present in the body. Rosemann has demonstrated that a diet deficient in chlorides leads at most to an insignificant reduction of the total chlorin content of the body in animals. Excretion of the element stops under such conditions, but signs of malnutrition are speedily elicited when chlorin is withdrawn from

the body by actual removal of the hydrochloric acid of the gastric juice through a fistula. Until losses are thus artificially enforced the gastric juice tends to maintain essentially its normal content of hydrochloric acid.

The ability of the animals, which Osborne and I observed, to continue in health for a time on a diet low in chlorin, might therefore have been anticipated; but, as we have remarked elsewhere, it could not have been expected that they would continue to thrive so long or attain so many times their original weight on such an extremely low chlorin intake. The outcome of these experiments cannot be due to substitution of other anions for chlorin but is attributable to a husbanding of this specific element. From the therapeutic standpoint the demonstration that a growing animal can fully supply from inorganic sources its requirements of the inorganic elements emphasizes anew that it is unnecessary to consider the presence of calcium, phosphorus and iron, for example, in natural foods to the degree that is currently believed.

Food and the Intestinal Flora.—There are times when the bacterial invaders of the alimentary tract give much concern to the gastro-enterologist. It is, of course, true that micro-organisms are always harbored in the recesses of the bowel, but the intestinal flora is by no means a constant one. On rare occasions specifically pathogenic bacteria are present. Under ordinary circumstances the types include more familiar putrefactive organisms, on the one hand, or acidophile bacteria, on the other. Herter and his associates were the first to demonstrate clearly the dependence of the types of bacteria developing in the alimentary canal upon the chemical character of the diet. In general it may be said that abundance of carbohydrates tends to favor the preponderance of the acid-forming types, whereas proteins permit the appearance of putrefying bacteria. Thus one may recall Torrey's observations on the effect of various high-calory diets upon the fecal flora of typhoid fever patients. It was shown by him that with some cases if lactose were added in amounts of 250 to 300 grams a day to the other ingredients of the Coleman-Shaffer diet there resulted a transformation of the fecal flora from the ordinary type to one strongly dominated by *B. acidophilus*. As an added illustration of the fundamental way in which the types of bacteria vegetating in the intestine can be controlled by the

chemical character of the food ingested I shall quote from the more recent studies by Torrey.⁸

"In feeding experiments with dogs it has been shown that two carbohydrates, lactose or dextrin, when added to a meat and rice diet caused such a marked development of aciduric bacteria of the *B. acidophilus* type that they completely dominated the fecal flora and effected the almost complete suppression of proteolytic types commonly found in the canine intestinal tract, even including *B. coli*. This purely fermentative flora would, furthermore, persist as long as the diet was continued, there being no tendency to reversion to the so-called normal flora. *B. bifidus* sometimes increases greatly under these dietary conditions, but generally was soon overgrown and suppressed by *B. acidophilus*, and, in fact, very rarely became the dominant type. A diet of bread and milk, which naturally contains both lactose and dextrin, was also followed by the establishment of a fecal flora consisting almost entirely of *B. acidophilus*."

Again, Torrey writes:

"Starchy foods all tended to effect a simplification of the intestinal flora and an elimination of obligate putrefactive bacteria. These foods with a large starch content differed in some degree in their efficiency as transforming agencies. White bread, potatoes and beans all tended to bring about a predominance of *B. acidophilus*, whereas rice proved rather less effective as an anti-putrefactive agent."

And of the protein factor Torrey has this to say:

"Various proteins were found to differ radically in their effect upon the intestinal flora, depending upon their source. Of the varieties tested the proteins of mammalian tissues were the only ones which markedly encouraged the growth and activity of the obligate putrefactive bacteria within the intestinal tract. A diet of fish brought to development a flora which was entirely different from that appearing in association with the feeding of beef hearts. Spore-bearing bacteria did not appear in the fecal specimens in more than insignificant numbers. There was a notable absence of the *B. welchii* types which constitute so large a part of the flora in connection with a meat diet. On the other hand, bacteria of the *B. coli* and *B. proteus* types were strongly predominant. Milk casein as an article of diet exhibited far less tendency to give rise to intestinal putrefaction than did meat

protein. . . . Vegetable proteins stand in strong contrast to animal proteins, especially meat, in that they do not offer the slightest encouragement to the growth of intestinal putrefactive types of bacteria. In fact, with a bread containing a very high protein content with a minimum of carbohydrate as marked an overgrowth of aciduric intestinal bacteria occurred as was observed in connection with diets to which considerable amounts of lactose or dextrin had been added."

It is needless to multiply quotations. They are concordant in suggesting that diet can probably be made a more potent factor than are ingested bacterial cultures in regulating putrefactive conditions in the bowel. The determination of the conditions under which desired intestinal floras can be established opens a fruitful field for the clinical investigator.

Dietetics offers no cure-alls to the gastro-enterologist, nor does any other mode of therapy. Calories, proteins, vitamins, acidophilic bacteria—these merely furnish viewpoints from which important problems of practice may be examined. The student of foods and nutrition is wont to be reminded that "a little knowledge is a dangerous thing." This is doubtless true, but I cannot gain the conviction that utter ignorance is a safety device. My sole justification for presenting a consideration of some food factors in nutrition has been the hope that the fragmentary items hastily reviewed may perchance suggest new possibilities in the practice of gastro-enterologists. Huxley once wisely remarked that science commits suicide when it adopts a creed. The open mind continually receptive to new suggestions guarantees a more healthful life.

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RECENT ADVANCES IN GASTRIC PHYSIOLOGY.

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In the lower animals and in embryos the gastro-intestinal tract is a simple tube from pharynx to anus. The stomach is formed by the bulging and bending of this tube and by the addition of one or more cecal pouches. In a 10 mm. human embryo the stomach is made up of three parts: the expanded lower end of the esophagus, the long tubular antrum, almost indistinguishable from the duodenum, and a small fundus. The end of the esophagus meets the pyloric antrum at the incisura angularis. Later, the fundus grows at the expense of the other parts so that in the adult the pyloric antrum is comparatively small, and the end of the esophagus is represented only by the cardiac antrum and a prolongation along the lesser curvature which forms the gastric canal.¹ Remnants of the "primitive tube" must then be looked for along the lesser curvature. It is interesting that this region is lined by a mucous membrane very similar to that in the bowel, whereas in other parts of the stomach we find the highly differentiated acid-forming cells.²

Now, there is considerable evidence for the view that the rhythmicity of the primitive gastro-intestinal tube was graded downwards from pharynx to anus much as the rhythmicity of the primitive heart tube is graded from the venous to the aortic end.³ During its development the heart tube is twisted on itself and develops bulgings where the future auricles and ventricles are to be. In these regions the muscle is specialized so that it can contract more quickly; it becomes more like striated muscle and in so doing, it loses much of its original rhythmicity.⁴ Through all these changes, however, a small strip of primitive highly rhythmic tissue remains, with its venous end still acting as the pacemaker. Similarly in the stomach we shall see that specialization in function has been accompanied by a specialization in the muscle and a loss in rhythmicity. As we should ex-

pect, the most rhythmic muscle is to be found along the lesser curvature, with what may perhaps be likened to a pacemaker at the cardia. Although the analogies are close, to avoid confusion it must always be remembered that the functions of heart and stomach are very different. In one the impulse spread so rapidly that the organ appears to contract as a unit; in the other a series of waves travel slowly over the sac, gently kneading its contents.

Differences in rhythmicity were shown by cutting off little strips of muscle from different parts of the stomach and getting them to contract in warm aerated Locke's solution. The segment from the lesser curvature next to the cardia always showed the greatest tendency to rhythmic contraction. Strips from the greater curvature and particularly from the pyloric antrum were slow in starting and many would not contract at all. The rate of contraction is fastest at the cardia and it decreases progressively to the pylorus. The amplitude of contraction was small near the cardia and along the lesser curvature. It was larger in the pre-antral region, and largest in the antrum. These peculiarities were found not only in various animals, but also in the stomach of an executed criminal obtained a few minutes after death.

If we watch the human stomach with the fluoroscope the contractions seem to begin on the greater curvature near its middle or a little below. As they approach the antrum they deepen, and suddenly their whole character changes. The indention becomes equally deep on the two curvatures so that the wave may cut the stomach shadow almost in two. It seems to me that these local differences in the peristaltic wave correspond perfectly to the regional peculiarities of tone and amplitude in the muscle through which it must pass. Serial X-ray plates show that the waves do originate near the cardia but that there they are very shallow.⁵ It is probable, then, that the waves which seem to begin in the lower third of the stomach have come as ripples from the highly rhythmic region on the lesser curvature near the cardia.

Although the direction of peristalsis may be maintained by or due mainly to the gradient in rhythmicity, other important and closely related gradients can be demonstrated. In the first place the region around the cardia is the most irritable part of the stomach. It also has the shortest latent period.⁶ The latent period is graded from one end of the stomach to the other. The

importance of this gradation is shown best in the simple stomach of the frog. There, an electric stimulus to the pyloric antrum will show itself as a contraction in from 13 to 14 seconds. Before that time elapses, however, the stimulus spreads to the cardia where after 8 seconds it shows itself as a peristaltic wave. This wave gets well down towards the pylorus before any change is visible at the point stimulated. We see then that this mechanism alone could insure the aboral direction of peristalsis.

The same gradation in latent period was shown in the excised strips of muscle when they were stimulated in a warm moist chamber.⁷ This study again brought out marked differences in the characteristics of the tissue from different parts of the stomach. The cardiac muscle was very delicate and suffered a great deal from the trauma of excision, handling and stimulation. The antral muscle, on the other hand, was tough and would react well after much trauma. It reacted even better after it has been in the icebox for 48 hours than it did at first. The cardiac muscle often would not react at all after such treatment. The shape of the contraction curve on the smoked drum was different and characteristic for different regions of the stomach. The muscle from the fundus showed a great tendency to remain tonically contracted after stimulation, while the antral muscle relaxed promptly. The pyloric antrum seems to be particularly fitted to carry on the active muscular work of the stomach while the fundus serves to maintain a steady tonic pressure on the contents. The muscle from the antrum has a color different from that in the rest of the stomach; it is redder and tougher like a gizzard. The muscle in the pacemaking region is the softest to the touch.

UNDERLYING METABOLIC DIFFERENCES.

As one after another of these regional differences came to light it seemed to me that there must be underneath them all differences in chemical structure and in the rate of metabolism. The first work on this problem was done on the small intestine.⁸ Little difficulty was encountered there in demonstrating a gradient of CO₂ production in the muscle from the duodenum to the ileum. Remarkably parallel to this gradient was a gradient of catalase content. Catalase is the ferment which liberates oxygen from hydrogen peroxid. A great deal of work has been done which

indicates that in some way this substance is involved in the process of tissue oxidation. Tissues with high rates of oxidation usually have more catalase per unit of weight than have tissues with slower rates of oxidation. It seems most likely then that the gradient of catalase content which Miss Starkweather and I have found in the wall of the stomach represents a gradient of metabolic activity.⁹ This gradient corresponds quite well to the gradient of rhythmicity already established. Furthermore, just as there is a sudden change from a low rhythmic rate in the antrum to a fast rate in the duodenum, so we find an abrupt rise in the catalase content as we cross the pyloric line.

So far very little work has been done on the anatomical side of the problem. Keith¹⁰ has found nodal tissue in the highly rhythmic area on the lesser curvature near the cardia. Openchowski¹¹ found peculiar ganglion cells around the cardia different from those in Auerbach's plexus. Schütz¹² and L. R. Müller¹³ have made similar observations. Although big histologic differences have been found in striated muscles and in the heart, very little attention has been paid to this point in the stomach and intestine. McGill¹⁴ found an embryonic type of smooth muscle persisting in some parts of the tract, but unfortunately does not remember just where these islands were.

More work should be done also on the pylorus. It is not generally known that there is a fairly complete connective tissue barrier between the muscle of the antrum and the duodenum. Ordinarily there are only a few bundles from the longitudinal coat which pass over.¹⁵ This explains the fact that the gastric waves do not run over onto the duodenum. Graphic records show that some influence may pass over to start peristaltic rushes in the bowel, but the deep waves, visible to the unaided eye, certainly stop at the barrier.

CLINICAL APPLICATIONS.

After years of experiment it has been established that the stomach can perform its functions quite satisfactorily after section of all extrinsic nerves.¹⁶ Now we see that these functions are probably dependent upon graded differences in rhythmicity, irritability, latent period and metabolism in the local neuromusculatory apparatus. If this is true, then any changes in these gradients must influence the peristalsis and the emptying time

of the stomach. Such changes have been found in sick animals. In distempered dogs and snuffling cats the gradients of latent period and metabolism were often reversed and practically always made irregular and less steep. This was due probably to the fact that the muscle at the cardia is so much more sensitive to adverse conditions and probably to disease toxins than is the pyloric muscle. Similar upsets were observed in the intestinal gradients in these sick animals.

The gradient might be reversed also by an irritating lesion such as an ulcer near the pylorus which could raise the metabolic rate in the surrounding muscle above that near the cardia. Such a reversal would probably give rise to the anastalsis which is seen sometimes with the X-ray.

A number of surgeons have commented on the fact that a V-shaped excision of an ulcer on the lesser curvature is likely to interfere with the emptying of the stomach while a sleeve resection usually gives a much better result.¹⁷ We can probably explain that now in the following way: Ordinarily the wave originating at the cardia has to travel faster along the greater curvature than along the lesser curvature because the greater is longer. Apparently both sides of the wave must reach the pylorus at the same time in order to secure good emptying. Now, time and time again while watching the human stomach under the screen I have seen the two sides of the wave reach the antrum unevenly so that they met not at the pylorus but at a point on one side or the other. Sometimes this was due to an ulcer on the lesser curvature, at other times I could not see what had interfered with the conduction. Sometimes the arrival of part of the wave at the pylorus seemed to block the other part of it still advancing along the greater curvature. It seems to me probable that such differences in conduction would be still more exaggerated after an operation which shortens the lesser curvature. Under these circumstances one side of the wave might reach the pylorus long before the other. Theoretically a sleeve should be removed with a longer side on the greater curvature than on the lesser.

While watching gastric peristalsis in the rabbit and also in man it has often seemed to me that there must be a wave of excitation traveling ahead of the visible contraction. Owing to its greater irritability and shorter latent period the pyloric ring responds to the excitation a little ahead of time. The resulting

contraction blocks the advancing peristaltic wave and keeps it from putting any pressure on the sphincter. This may explain why some stomachs do not empty well in spite of good peristalsis and a patent pylorus. Time and again in cats I have watched a similar mechanism at work at the ileocecal ring enabling it to contract firmly in the face of advancing waves, protecting it from being forced, and blocking the reverse waves before they could bring much pressure to bear at that point.

While peeling off the strips of muscle it was found that the submucosa was quite deficient along the lesser curvature from the cardia to the antrum. The mucosa is fastened to the muscle there much as the skin is to the palmar fascia of the hand. In the rest of the stomach and particularly in the antrum the strips could be peeled off easily. These differences may have some influence on the spread of carcinoma and on the penetration of ulcer.

It is an interesting observation that in animals the region corresponding to the duodenal cap has a poor rhythmicity. Excised segments put into warm oxygenated Locke's solution often do not contract at all, and when they do, they generally show an irregular rhythm with small amplitude. This may be due partly to the peculiar festoon-like arrangement of the muscle fibers in this region. As would be expected, this comparatively inactive segment, situated as it is between two strong and active ones, tends to remain filled with food for long periods of time.

It is a well-known fact that carcinoma very rarely originates in the duodenum and when it begins in the stomach it will not spread across the pyloric line. It seems probable now that this is due to a difference in metabolic rate on the two sides of that line. Estimations of the catalase content of the mucous membrane from different parts of the digestive tract showed that the lowest figures are to be obtained in the pyloric antrum and the highest in the duodenum.¹⁸ Now, Child¹⁹ has shown with simple forms of life that the embryonic, undifferentiated and rapidly growing cell loses its capacity for growth as it fills up with the comparatively stable structural substances which are acquired during differentiation and specialization. This process leads to senescence and death. After the metabolism has become greatly slowed these cells may suddenly begin to use up this metaplasia; they de-differentiate and re-acquire the faculty

of growth. Goodpasture²⁰ has recently pointed out how this mechanism, so useful in the rejuvenation of simple forms of life, can threaten the existence of the higher forms. It may be, then, that that part of the gastric mucous membrane with the lowest metabolic rate is, as it were, most senile and therefore most subject to cancer. Tumors originating there cannot spread into a mucous membrane which is kept young by its high metabolic rate.

One other thing might be mentioned in closing, and that is the apparent uselessness of gastric faradism as a therapeutic measure. I found it impossible sometimes to get contraction of the gastric muscle when strong currents were applied directly to the serous coat. This was observed not only on animals opened under an anesthetic but also at operations on men and women. Meltzer²¹ found many years ago that it was almost impossible to stimulate the muscle through the mucosa. Even when the current gets through there is no reason to hope that it will act so appropriately as to restore the downward gradients when they have been altered by disease.

SUMMARY.

The gastro-intestinal tube may originally have been constructed so that the rhythmicity of any one segment varied inversely as the distance from the pharynx.

The stomach has been evolved from this simple tube much as the heart has been enlarged and specialized. The remnants of the primitive tube are to be looked for along the lesser curvature.

A highly rhythmic area has been found on the lesser curvature near the cardia.

Strips of muscle excised from different parts of the gastric wall can be made to beat rhythmically in warm aerated Locke's solution. Speaking roughly, the rate of contraction varies inversely as the distance from the cardia.

Differences in the depth of the waves as they sweep over the stomach can be explained by differences in tone and amplitude of contraction of the muscle in different parts of the organ.

There is a gradient of irritability from cardia to pylorus. The latent period is shortest near the cardia and longest in the pyloric antrum.

The muscle in the pars pylorica appears to be quite different

from that in the rest of the stomach. It is especially fitted to do the hard work of that organ.

The pyloric ring is more irritable and has a shorter latent period than that of the pyloric antrum. This may have considerable bearing on the problems of poor gastric emptying in ulcer.

The duodenum is much more irritable than the antrum and its rhythmic rate is much higher.

The muscle on the lesser curvature near the cardia is very sensitive to trauma and to adverse conditions which seem to have no effect on the muscle from the antrum. This difference may account for the fact that the normal downward gradients of rhythmicity and latent period were altered and even reversed in sick animals.

Evidence is presented which suggests strongly that there is a gradient of metabolism underlying and perhaps giving rise to the gradients of irritability, latent period and rhythmicity which, it is believed, determine the direction of peristalsis. The metabolic gradient is often found reversed in sick animals which are refusing food.

There is a gradient of metabolism also in the mucous membrane of the stomach. The lowest values in the whole digestive tract are found in the antrum. It is shown how this may explain the high incidence of cancer in that region and the inability of the tumors to cross over into the duodenum where there is a very high metabolic rate.

An explanation is given for the fact that a sleeve resection gives a better functional result in ulcer of the lesser curvature than a V excision.

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DISCUSSIONS.

DR. HEMMETER: I wish to express my admiration of this very beautiful biologic research on gastric peristalsis and secretion. You are aware that Dr. Alvarez has emphasized that the entire fundamental peristalsis is myogenic, with that he puts himself in line with the physiologists who hold that the heart's contraction wave is myogenic in opposition to those who think that the heart contraction wave is neurogenic. That subject is not closed. The analogy and comparison of the peristaltic wave of the stomach with that of the heart is not felicitous. If you cut out the sino-auricular node from the heart of a Selachian, for example, the rhythmicity stops gradually; there is no autonomic beat any longer after that. The only contraction wave you get after that in cold-blooded animals is by stimulation. I am speaking only now of cold-blooded animals. It is only with the heart of the shark or the amphibia that such excision of the sino-auricular node (observations of the English observers Keith and Platt Flack) can be excised with success. There is this essential difference between heart contraction and gastric peristalsis, the rhythmicity and automaticity can be resumed spontaneously in the stomach, even after surgeons have removed that part of this organ in which the peristaltic wave apparently starts. But in the heart of the cold-blooded animals the contraction wave from the sinus venosus over the auricle to the ventricle, ceases after the sinus venosus is separated from the auricle and ventricle by a ligature (first ligature of Stannius). The so-called second ligature of Stannius may call forth the contractions of the ventricle again, but such contractions are not normal nor do they persist long and their occurrence has given rise to various interpretations, mostly hypothetical.

DR. BASCH: The question of excision (v-shaped excision or sleeve resection) in chronic gastric ulcer interests me. It would be interesting from the clinical standpoint to follow out the practical results

obtained. We must leave to those who have time to do laboratory work the theoretical working out. From the Mt. Sinai Hospital, Drs. Berg and Wilsinsky issued their researches, from whence comes forth the dictum that the v-shaped excision is a detrimental thing; that the intrinsic nerves of motility are located in the lesser curvature, and therefore the v-shaped resection causes a paresis with a resulting hour-glass contraction. As a matter of practical result, I have had this v-shaped resection done by others, and I wish to quote especially one case where the v-shaped resection was done a year and a half ago. This patient had a long history of ulcer pains and so on, and as was demonstrated by the X-ray and proven by operation, a chronic penetrating ulcer of the lesser curvature. Every two months for one year I kept X-raying this patient after his operation. There was complete restoration of stomach outline, perfect health and perfect motility as far as one could tell from the X-ray and from laboratory tests. Now, it would be interesting for us gentlemen, who are brought into clinical contact with our patients, to take a definite stand upon this question. Undoubtedly we get good results from sleeve resection; but the sleeve resection is a very severe traumatism to the patient. It is an operation to be done only by the most skillful surgeons, and as an operative choice we would favor a v-shaped resection if the views of Berg bearing upon this form of resection are not borne out by the rest of you. I do not know whether Dr. Lichty in his discussion of influenza and gastric conditions is going to talk about the point brought out by Dr. Alvarez's paper, namely, that the toxin affects the smaller curvature near the cardia, and as this is the less resistant portion, we therefore might have a number of our clinical manifestations. I have had occasion to see a number of cases that were diagnosed as ulcer following influenza and in no case was I able to bear out a diagnosis of ulcer.

DR. KAST: I wish to voice our appreciation of Dr. Alvarez's paper. It is one of the most interesting presentations which has come before our association, and we are very grateful for his bringing it before us. About two or three years ago I made a series of investigations as to the effect of fatigue upon the gastric peristalsis and presented the result before the Society for Experimental Biology and Medicine of New York and came to the conclusion that in a number of cases we could not find any definite relation between fatigue and gastric motility as tested with the stomach tube and with the X-rays. But later the same subject was taken up again and was studied on a larger material selecting cases which were found to be sensitive to the effect of fatigue. In these cases we soon realized that there were two parts in the digestive tract which seemed to be particularly susceptible to the effect of fatigue, namely the stomach and the caecum and ascending colon. In these two parts we get definite retarding effect of fatigue upon motility.

Since fatigue has been drawn into the field of chemical research as

a problem of metabolic disturbance, it brings it very close to the line of investigation to which Dr. Alvarez has referred today; and since we know that a number of so-called gastrointestinal neuroses are at least closely related, if not caused by chronic fatigue, physical or emotional, we have here three groups of facts which may find a common explanation.

DR. MILLS: Dr. Alvarez's address was very reassuring to me. You may recall that last year I presented a study of gastric motility as studied by the X-ray. The barium meal has been criticized as being artificial. Of course, we cannot say that conditions occurring after a barium meal are the same as occur after an ordinary meal; at the same time it seems to me that the burden of proof is to show that similar conditions do not occur. The essayist incidentally remarked on pyloric spasm. After this year's work I will almost go so far as to say that there is no such thing as pylorospasm, except possibly where a lesion directly involves the pyloric sphincter; and I have made sincere attempts to determine what influence it may have in impaired gastric motility.

With reference to the influence on gastric motility of v-shaped and sleeve resections we must individualize. The surgeon knows that in many cases if he does a v-shaped incision, and does not do a gastro-enterostomy, his result will not be good. The gastro-jejunostomy is a drainage operation which saves the day for the surgeon by compensating for the damage done the motor capacity of the stomach. Certain types of low hypotonic stomachs will not drain well after a v-shaped or sleeve resection without a gastro-jejunostomy. Other opposite types of high markedly tonic stomachs will. Gravity is an important factor in the emptying of the stomach. The most favorable results in gastric surgery occur in two conditions. Where gastro-jejunostomy is done for high-grade peripyloric stenosis and in cases in which the stomach has been largely resected and gastro-jejunostomy done. In the case of resection the impaired motor functions, the result of mutilation with consequent disorganized peristalsis, is compensated for by diminution in gastric capacity and gravity. In other words, gravity plus an artificial hypertonus, the result of the limited capacity of the small gastric sac left, furthers motility.

DR. J. A. LICHTY, of Pittsburgh: I do not expect to add anything to the discussion of this paper; only to express my gratitude to Dr. Alvarez, who has pointed out to us that there is probably a "gastric pacemaker," and that he has located this. When the electrocardiograph came into vogue and the cardiac problems began to unravel, I felt, from the gastro-enterological side, we were compelled to take a back seat, because there were so many things that we could not explain so definitely, as were those being explained in the heart. I am very glad that Dr. Alvarez has brought out the comparison between these two organs. In fact, I have said to those students with whom I happened to be associated that I believed the time would come

when we would have a gastric pacemaker which would explain gastric phenomena just as easily and just as nicely as the pacemaker of the heart explains the phenomena of the heart.

DR. ALVAREZ, closing: Just as the impulse may originate in other parts of the heart when the sinus node is damaged, so the waves in the stomach may begin elsewhere when the usual place near the cardia is diseased or removed. In a distempered dog you can show that the gradients are flattened or even reversed, and I believe this has something to do with the fact that they refuse food and retain in their stomachs for hours that which is forced on them. It is interesting that drugs like digitalis and chloroform which tend to upset the heart gradient also tend to upset the intestinal gradient.

In fatigue neuroses of the digestive tract the surest way to restore the normal gradients and to bring relief from symptoms is to insure the patient plenty of sleep by giving sedatives at night.

NORMAL AND PATHOLOGICAL GASTRIC FUNCTION.

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The study of gastric function demands revision. It is essential that before we attempt to make progress we have clearly in our minds the fundamental principles which dominate the problem. The roentgenologist has made such strides that the form, position, motor function, and changes in morphology are readily appreciable, but it behooves the clinician to keep pace with these advances by formulating the principles underlying normal gastric secretory work. The literature is full of painstaking labor with endless pages of so-called gastric analyses portraying a given series of findings associated with certain disease phenomena, but it is remarkably devoid of accurate observations on the untraumatized individual in health. Before any conclusion can be drawn, we must have a series of standards for comparison, and we must not fall in that pitfall which has beset so many investigators to attempt pathological deductions in the absence of accurate normal data. How often have I heard the statement that gastric analyses with the exception of a few well marked conditions were valueless, and I must confess that the utter obscurity surrounding the interpretation of any finding might well engender such remarks. Let us therefore start with certain facts and attempt to build an edifice which, however frail, at least represents an attempt to throw daylight on the problem.

In the first place, gastric analysis is a measure of function. It is a functional test, but unlike other organs whose output under various conditions is more or less constant, we are compelled to acknowledge that gastric function consists of a series of rhythmically recurring cycles in response to a definite stimulus or series of stimuli. This immediately introduces the factor of variability, that is to say that the sum total of the gastric contents both physically and chemically is undergoing a constant change which is readily proven by fractional examination. The latter method of procedure immediately demonstrates that any given figure or series of figures cannot be accepted as an index

of gastric function. I would, therefore, propose in order to bring some degree of uniformity that we take those essential facts which physiologists and clinicians have demonstrated as characteristic of normal digestion and attempt to define the characteristics and limits of normal gastric digestion as a basis for the study of pathological deviations. In this way alone can we hope to achieve progress. I will therefore enumerate those points which might serve as a basis of normal gastric digestion and as a means of determining pathological deviations:

(1) Normal gastric digestion is a series of cycles. In other words, there is a rhythmic alternation of periods, which for the want of a better name I have called digestive and interdigestive periods. These cycles follow each other regularly and are distinguished by differences in almost every essential particular. The quantity, total and free acidity, enzyme value, trypsin content, N content are different. The interdigestive period is the period in which the so-called gastric residuum on the "fasting" or "empty" stomach is obtained, but we have been able to demonstrate that the stomach is never empty. Bergeim, Hawk, and myself have defined the characteristics of this interdigestive period in 100 normal men, Fowler from our laboratories obtained the same data at Ames, Iowa, on eighty normal women and the data are sufficiently similar to enable us to form conclusions regarding this period in health and its deviations in disease. These two periods, the "digestive" and the "interdigestive" periods, represent simply the response of the stomach to perfectly correlated or co-ordinated stimuli, one or many of which may be disturbed in disease. The enumeration and nature of these stimuli is a work which the physiologists have largely contributed to and one which bids fair to enter the domain of clinical medicine.

(2) Normal secretory digestion follows an absolutely characteristic course in each individual, so that over a thousand curves obtained from healthy men, the individual always reacted in a definite sort of way highly characteristic of the type. We therefore went on record as distinguishing three types of normal human "digestive" period, one over 40 per cent. in which the response was accompanied with a pronounced outpouring of secretion, and high acid figures, which we called the "hyper-secretory type," another with low general output which we called

the "hyposecretory type," and finally a third following our pre-conceived notions on the subject which we called the "isosecretory" type. We are ready to substantiate our original findings, which will all eventually be published, by further observations which make it clear that these types are true types and must be considered before any pathological interpretations be drawn. A single instance, a known "hyposecretory" type, will be found to manifest "hypersecretory" findings and invariably there is a disturbance in gastric digestion. The findings of high acid figures, and increased acidity in health such as described many times in the literature as characteristic of ulcer, peritoneal inflammations, chronic appendicitis, etc., immediately demonstrates that this information is both wrong and misleading. Disease consists in reversal of form or deviations in type or exaggerations in tendency. The effect of fatigue, exertion, time of the day, must be more accurately outlined in these studies.

(3) The components of the normal digestive response must be defined. It is time that not only the physiologist speaks of the "psychic" or "appetite" secretion, and the chemical secretion. If we do not make a move to accept these findings, we shall find ourselves being compelled to make such a move. We have measured the appetite secretion in health with a "psychic test" meal—namely, the cooking and chewing of 100 grammes of meat, a procedure almost as simple as the daily test meal. It is clear that on average as much as 240 c.c. of gastric secretion of general high acidity is obtained. It is equally clear from a limited number of observations that in disease this psychic secretion is altered. In one case we obtained in ulcer over 300 c.c. In one case of my own of gastritis, I was able to demonstrate a psychic secretion markedly below normal. Therefore, a whole chapter remains to be written about psychic secretion in health and disease. We have shown how simple such tests are. Why not measure their aberrations? We demonstrated the effect of different psychic stimuli as well as atropine on this secretion. Again it is possible to measure the "chemical response" of food by injection through the tube, while holding the tube behind the individual. Such a secretion starts early and not late, as would be supposed. Therefore, such a meal or any meal introduced through the tube might be called for the want of a better name the "chemical test"

meal. Composing it presumably are simply the secretions in response to chemical stimuli.

(4) The next point is the tendency of the healthy stomach to reduce all substances to a fixed point. I wish to refer to a diagram which emphasizes this extraordinary elasticity of the stomach to the most diverse stimuli reducing all to a more or less constant level. In this we demonstrated the fact that acids were neutralized to a minimum, that soluble alkalies were neutralized and followed by secondary stimulation, that concentrated salt solution and dextrose solution for a while inhibited secretion only to be followed after a while by this same secretion of more or less constant level. THIS LEVELLING PROCESS IS THE MOST CHARACTERISTIC THING OF THE NORMAL STOMACH. FOODS MOST DISSIMILAR IN APPEARANCE, FOODS BOLTED AND CAREFULLY CHEWED, MILK RAPIDLY SWALLOWED AND SLOWLY SIPPED, VEGETABLES OF THE MOST DIVERSE NATURE ARE ALL SUBMITTED TO THIS LEVELLING AND NEUTRALIZING INFLUENCE SO THAT DOUBLE INTUBATION WITH A TUBE IN THE STOMACH AND ONE IN THE DUODENUM INDICATE THE ENORMOUS AMOUNT OF WORK DONE BY THE STOMACH—MOTOR, SECRETORY AND ELIMINATORY. All substances begin to lose their identity and are gradually converted to the eventual equilibrium point after which the stomach slowly revolves back to the "interdigestive" phase. Therefore, the most important single characteristic of the normal stomach, a characteristic lost in many diseased conditions, is the ability of the organ to reduce substances of a most diverse nature to a state of more or less equilibrium, within a fixed time limit.

(5) The factors concerned in this reduction are not merely the elaboration of the secretion but control of the secretion. Some time ago we pointed out the intimate association of the pancreas and the stomach, in confirmation of Boldyreff's theory of the autoregulation of gastric acidity. It is a fact readily proven by careful study that the duodenal secretion is disassociated and there is regurgitation of pancreatic secretion, by which this is regulated. This factor must be crippled in disease and from a limited number of cases there is undoubted crippling of this normal function. In some cases in disease it does not

occur. In many of the low acidities the low acidity is due not so much to poor output but to a high and persistent pancreatic backflow, making the statement of one observer "intestinal digestion" in the stomach a truism. Here, then, is another chapter for investigation. One which is full of important data, not the least of which is the association of the pancreatic reflux with the phenomena of the interdigestive phase. Disturbances in this "equilibrium" factor which depends on the perfect co-ordination of all stimuli are too numerous to mention. They occur in almost every case of gastric disease and functionally in all cases. Disease upsets all the mechanisms, in one case the secretory, in another the pancreatic, in another the motor mechanism, but the regular rhythmicity of function is lost.

(6) *Gastric Acidity*—Probably more has been written about gastric acidity than any other single point and yet many other points are equally significant. Gastric acidity is but one of the elements in the interpretation of gastric curves. But gastric acidity in the light of our studies has emphasized a series of facts which I attempted to discuss at the last meeting. I made the statement that there is no level of titratable acidity in disease that has not its equivalent in health. I still maintain this is true in 40 per cent. of our cases. In health we got figures which in all text books up until that time had been described as those of hyperacidity. Low figures are far more significant. This question only needs reiteration; my standpoint today would be that at certain phases, the interdigestive, the beginning of the digestive and the end of the digestive period high acid figures are abnormal, but to the student of gastric physiology the most protean manifestations can occur. High, unsaturated free acid with obvious disturbances in acidity, is a pathologic phenomenon almost always associated with aberrations in the quantity of secretion rather than acidity titre. Therefore, the question is not how high is acidity, but, more important, at what points is it high? I have refrained from writing about curves in disease, although I have a great many. My conceptions of alterations in disease will follow briefly. REMEMBER, HOWEVER, THAT THE FIGURES WILL CONSTANTLY VARY AND NO ONE EXAMINATION WILL DETERMINE THE ACME OF GASTRIC ACIDITY. FURTHERMORE, THERE IS NO ASSOCIATION BETWEEN THE SUBJECTIVE FEEL-

ING OF HYPERACIDITY AND THE TITRATIBLE ACIDITY. ALL OF US HAVE SEEN IT IN HYPOACIDITY AND EVEN IN ACHYLIAS.

(7) We must define the secretory output in health. Measuring the total volume is no satisfactory method. The velocity of secretory output changes. A comparison between food volume and secretory output is arbitrary. The stomach may speed up its secretory output at any time. I know only one positive way and it was the method employed in our "psychic" studies in which during the continuance of psychic stimuli the stomach was completely emptied at intervals. A rough approximation may be formed, by comparison of relative food volumes, but only an accurate determination of the secretory output will actually determine secretory velocity. Carlson has done it on his fistula man, but no figures on traumatized individuals can be accepted as those of men in health. In disease again, every variation can occur.

(8) A sharp distinction should be made between the components which are associated with acidity and other components of the gastric juice. The enzyme content is open to many variations dependent on fatigue. Here again we must accept for the human organism the laws laid down by the physiologist and accept deviations of these laws as deviations from health. The enzyme content in the interdigestive phases of gastric function are highly interesting. For instance, there was a relationship between pepsin and low acidities, which disappeared on high acidities. In one of our cases, disassociation occurred from fatigue, the ability to curdle milk being lost, to be regained later on.

(9) Another important point in the secretion is the presence of various substances such as soluble albumen, the amino acid content, as determined by the Formol index of Sorensen. Their curve follows certain definite standards in health and the diseased variations are rather clearly defined. Amino acid follows closely combined acid curve.

(10) Finally, a test meal is merely a measure of gastric function. The number of test meals is legion. Their function is simply the mensuration of gastric capacity and work. Complexity in the composition multiplies the confusing factors in interpretation. Simplicity is obviously the desirable factor. The

water meal is the simplest test meal, because every added factor is clearly shown, but its stimulatory properties are not maximal nor is it possible to clearly delimit the end point.

The presence of the clearly pathological factors such as pus, blood, albumen, increase in mucus and cytology are best shown in this way, and here its value stops. For the chemistry of the mucosa and the exudative factors seen in the various catarrhs and in gastritis, and carcinoma it is unexcelled. For general routine work, we have tried every variety of meat, vegetables, cereals, fruit, and none of them for practical purposes are superior to the Ewald meal. The protein content is more or less constant, the fat is negligible, and the discordant factors are reduced to a minimum. Meats and fish produce the highest acidities, fruits and green vegetables the lowest. A solution of peptone will answer for protein, dextrose in concentrated form will inhibit, in dilute solutions will be followed by stimulation. An attempt likewise can be made to measure the neutralizing power of the stomach 100 c.c. of 5 per cent. sodium bicarbonate being neutralized in 30-40 minutes by many normal stomachs. All these things are possible, but they simply multiply the problem rather than simplify it.

In enumerating, therefore, the characteristics of normal gastric function, we may say that an enumeration of the above factors will give us a tangible basis for the determination of diseased factors. It emphasizes the great elasticity and adaptability of the normal stomach, the rhythmicity of its action, the sequence of its components, the factors concerned in its evolution, and, finally, the variability of its manifestations. Briefly reviewed, they are (1) a series of cycles, with definite digestive and "inter-digestive" phases; (2) the constantly changing character of the digestive phase, until the equilibration point is reached, highly characteristic for each individual and divided into three normal types, all of them evolving within definite time limits; (3) with highly characteristic interdigestive phase with certain definite limitations both as to quality and quantity of its ingredients in health; (4) a delimitation of the digestive phase into readily recognizable "psychic" and chemical factors, either of which or both are modified in disease, the essential nature of the healthy secretion being already defined; (5) the tendency of the healthy stomach to reduce all substances to a point which might be called "gastric equilibrium" preceding final evacuation, this levelling property being lost for various substances in disease; (6) gastric

acidity which evolves in definite fashion in health reaches certain heights for both the digestive and the interdigestive periods, undergoing every variation in disease; (7) the fact that gastric acidity in health may reach any figure found in disease so far as titratable acidity is concerned and the recognition of the fact that figures which are characteristic of so-called hyperacidity are found in health; (8) the recognition of automatic regulation of acidity pancreatic secretion as a normal phenomenon distorted in disease; (9) the recognition of the true importance of the acid content, enzyme content, amino acid and soluble albumen content, and the relative importance of frankly pathological substances such as pus, blood, mucus, bacteria. When these factors are clearly defined, as clearly defined as are the morphology and motor phenomena of the roentgenologist, we shall make positive progress in gastric disease. The effect of these studies reveals much that is absurd in gastric disease and attempts to throw light on this problem. Regarding all the above points, we must have irrecontrovertible data.

Disease reverses these findings. It alters the sequence of the periods. Ulcer first alters the motor and secretory balance, but may evolve with but little evidence of trouble, sooner or later, the vicious cycle of motor delay, infringement and destruction of the interdigestive period, delay in equilibrium point, starts a vicious cycle in which finally all semblance of gastric sequence is lost. In the chronic inflammation the mucosal reaction or response is slower, the cycles are abortive and incomplete, digestive work is insufficient. In atony and dilatation the normal interdigestive periods are reduced or lost with eventual tendency toward the total loss or disruption of function. Acute inflammations declare themselves not only by intolerance but to a total distortion of gastric sequence. Cancer being a mucosal disease, distorts the cycles and if associated with pyloric block, destroys the interdigestive phase, in any event both phases are lost, and from rhythmic sequence we may consider the stomach a rigid tube. Not only do all these gastric disturbances alter the normal evolution of digestion, but every factor connected with digestion can be altered. Toxins, chemical or bacterial, from altered metabolism or focal infection, can profoundly modify gastric digestion, psychic disturbance and nervous diseases as a group, play a definite rôle—the circulation and elimination—all of these can

and do alter this evolution of gastric digestion. This subject is too lengthy to discuss in this paper.

This brings us to the modification of gastric function in disease, a subject which is filled with promise but which is still awaiting solution. I have a mass of data which awaits study and interpretation, but the following points seem to me justifiable: (1) Alteration of gastric function can take place reflexly, through the nervous pathways, through the circulation, by direct mucosal changes, and finally by means of direct disease of the mucosa. An astonishing thing regarding ulcer is the fact that non-obstructive ulcer will give a series of curves closely approximating the acid curves in health, stenotic ulcer immediately throws out the co-ordinating mechanism, the pancreatic reflux, the pylorospasm, the reflex vagus phenomena resulting in vagotonia and the result is immediate a rupture of one or all of the points mentioned above. The interdigestive approaches the digestive cycle, the secretory velocity is increased, the pancreatic reflux is diminished, the chemical phase is exaggerated and prolonged, the equilibrium centre is indefinitely postponed, the gastric content varies in the direction of increased acidity and enzyme content, decreased amino acid content, decrease in trypsin. Cancer produces aberrations of an entirely different nature, by extension into the mucosa it destroys its action, by extension of infection it produces generalized gastritis, which result in mucosal deficiencies, reduction in acidity and enzyme output. It may or may inhibit the secretion by specific products, but in the beginning in many cases there is but little diminution in acid content; it adds its specific pathological exudates to the contents, the acid goes down, the trypsin and amino acid content up. Extragastric disease is distinguished from intragastric disease by the absence of the specific factors such as specific pathological exudates, amino acids, blood, pus, seen in gastric lesions. Gall bladder disease may act through associated duodenitis, producing gastritis, or through reflex agencies on the pancreas, but in early gall bladder disease, such as cholelithiasis without marked cholecystitis, the gastric signs are those of vagotonia, the secretory and functional imbalance is that of vagotonia. Abdominal adhesions, appendicitis, cholecystitis of the first grade, may depress gastric acidity through toxins, but more often their effect is by way of the vagus and sympathetics. Pus infection can reduce or totally obliterate gas-

tric sequence, infected tonsils, teeth—an exactly similar mechanism can come about through a metabolic disease such as gout or a deficiency disease such as pellagra, or a blood deficiency such as the profound anemias. Our business is to determine the effect of each of these on the two phases of gastric function. We can depress gastric secretion by white of egg, atropine hypodermically, concentrated dextrose solution, and yet the mechanism of their action must be different. Those of you who study pellagra should study the entire gastric sequence of the disease, the enzyme sequence, the tryptic content, and compare these with normal findings. The gastro-enterologist must be an internist first and a gastro-enterologist second. He must correlate his data. He must search for facts. No studies on the gastric analysis of ulcer have helped me unless I study the sequence of disease and compare it with the normal sequence. Why talk of hyperacidity? which means nothing. Of gas on the stomach? which is either aerophagia or only rarely fermentation. How are we to interpret gastric disease until we know exactly the etiology? Toxins upset autonomic balance, intestinal stasis alters the gradations of various portions of the gut according to Alvarez. Coated tongue, globus, heartburn, nausea and vomiting are merely due to mild reversed peristalsis according to this same author, but mild reversed peristalsis produces total distortion of the sequence of gastric cycles, the equilibrium point is delayed, the digestion prolonged, neutralization in many cases is altered, and not merely secretory but motor co-ordination is upset. Atony and dilatation destroy the interdigestive period. Rest relieves because it restores normal sequence.

And now we come to therapeusis. Therapeusis is successful as it restores gastric sequence and rhythmicity. Disease, whether toxic, mucosal, blood, direct lesions, alter sequence. **THIS IS THE FUNDAMENTAL KEYNOTE. THERAPEUSIS AIMS TO RESTORE THE REGULAR AND IN PROPORTION AS THIS NORMAL RHYTHMICITY IS RESTORED IS THERAPEUSIS SUCCESSFUL.** The slight delay and hypersecretion of ulcer with lessening of interdigestive period is shortened and the later period restored. Atony and dilatation, in which the interdigestive period is markedly shortened through rest and appropriate diet result in restoration of the interdigestive period. The exaggeration of secretory output extending beyond the di-

gestive period in hypersecretion is restored to normal only when it can be demonstrated that the cycles recur in normal manner, and so it goes throughout the line of gastric disturbances. THERAPEUTIC SUCCESS DEPENDS ON A RESTORATION OF THE ALTERED GASTRIC SEQUENCE BOTH SECRETORY AND MOTOR TO NORMAL STANDARDS.

DISCUSSION.

DR. HEMMETER: It certainly is a very paradoxical thing that we should get pancreatic secretion from the duodenum without bile, because the pancreatic duct does not open into the duodenum directly. It does open into the diverticulum of Vater into which the bile duct opens also. I don't wish to be understood as denying the observation, but only to emphasize that it is surprising that pure pancreatic juice without bile should have been obtained. I would like to ask Dr. Rehfuess whether he is sure that Pawlow said that the milk curdling effect of the gastric juice and the proteolytic effect of the gastric juice run different phases. If I remember Pawlow correctly, his great argument was that pepsin and renin are identical; that they are one and the same thing, and that he showed the milk curdling effect and the proteolytic effect ran almost parallel curves. I would like to know the further experiments of Dr. Rehfuess which led him to believe that the milk curve was an entirely different phase and ran an entirely different curve from the proteolytic effect of the gastric juice.

DR. SAWYER: I would like to ask in connection with this very interesting description of results, which are quite in accord with the clinical experience of most of us, I believe, whether or not in determining those superior curves the individuals as well as the diseases were studied by Dr. Rehfuess. In all the work which I did, clinically, I found a vast difference in people in their nervous reactions, in their susceptibility to peripheral stimuli: and it is a matter of common experience with me to find that the individuals who present just this superior type are individuals whose reactions, knee jerk, tremor and vasomotor phenomena, all are of a similar classification. I would ask if we should hold these simply to be the internal phenomena of the stomach or its muscular activity, or whether we should not also study the individual as to these general conditions. One person sluggish in reaction will not show this. The other person who has the general phenomena of excitability will show these phenomena. They are not uncommon. They are subject to easier fatigue than those individuals without exceedingly active nerve reactions. The whole individual must be studied, and these will be proved into the interpretation of a whole man, and diagnosed no different, whether a man is 300 pounds or 90 pounds in weight. He may be healthy, although he is far from

the average in either case; but we must bring these two into the universal study of the man.

DR. REHFUSS (closing): There is a definite tryptic content in the stomach. I have seen these experiments. I have seen the gastric contents, and I know there is a casein-reducing substance which comes into the stomach without bile, and which is found there. The only difficulty we have is in understanding how that reflux takes place. A fact is that a carbohydrate meal causes usually no reflux of bile; the bile usually occurs at the termination of the fat meal, and this trypsin is found in the stomach. It is in small amounts and gradually increases towards the end of digestion in many instances. The question of the parallelism of the pepsin and the acidity curve, and pepsin and renin: I do not intend to go into the discussion. Pawlow held that there was no parallelism as digestion proceeded. In the pepsin and in the acid curve we find that there was a parallelism in the elimination of pepsin and not in acidity. In other words, the pepsin went up slowly with the acidity; but when the acid reached a certain concentration, then the pepsin remained more or less a plateau. Regarding the question of individuals: We studied one hundred normal individuals, who are symptomless, in average health, following a definite line. We take a hundred medical students. I suppose not two of them look alike; but we take them because they have no gastric symptoms. So far as the acidity is concerned, it is not a question of the strength of acidity, but where the high acid figures occur.

EFFECTS OF RESTRICTED (SO-CALLED ULCER) DIETS UPON GASTRIC SECRETION AND MOTILITY.*

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Medical literature offers us several studies of the clinical end-results of medical treatment of gastric and duodenal ulcers. The chemism and physiology of the stomach after operation has also been studied. But practically no information can be obtained regarding the direct results upon the stomach of the various forms of medical treatment. Whether clinical improvement during an ulcer cure depends upon a change in the chemistry and motility of the digestive organ remained until recently a matter of speculation rather than a demonstrated fact. The most illuminating articles on this subject are those contributed by W. W. Hamburger¹ of Chicago. He was able to demonstrate during intensive medical treatment by radiographic means, the actual healing of ulcers, the disappearance of "niche" phenomena and the restoration of normal peristalsis and motility. While he refers from time to time to changes in the chemistry of the organ as a result of restricted diet, his attention is focused mainly on the radiographic picture of the healing ulcers.

J. Friedenwald, with Baetjer (*Trans. Assoc. Amer. Physicians*, 1903, p. 9) carefully observed radiographically the function of the stomach during medical treatment. His observations led him to conclude that several weeks, even months of protracted diet were necessary before the disappearance of signs of abnormal function such as exaggerated peristalsis, delayed motility, irregularity of contour, etc. The studies of Friedenwald are the first attempt directly to observe the effect of medical treatment on healing ulcers.

It is upon the chemism and the motility of the stomach while

*It is a great pleasure to acknowledge with sincere thanks the kind privileges extended by the Attending Physicians of the hospital in the course of this study.

undergoing medical treatment that I am attempting to contribute some facts. This paper is based on observations made upon 34 patients. The cases belong in one general group; they were either proven instances of gastric or duodenal ulcer or they were cases suspected of bearing ulcers. In five of the patients ulcer was demonstrated by operation at a later date; in 21 of the cases ulcer was the clinical diagnosis, based upon the history, physical findings, chemical data and radiographic evidence. In the remaining 8 subjects, the final clinical opinion was that they were suffering from a gastric neurosis or a functional disturbance of the stomach.

The symptoms which these patients presented in common were periodic attacks of heartburn, pain after meals, acid eructations, occasional vomiting, and loss of weight. Practically in all these cases hyperacidity was chemically demonstrable, hypersecretion was frequently present, and delayed emptying of the digestive organ was a prominent factor.

Since intra-abdominal diagnosis at its very best is conceded to be inaccurate and open to error, it is better in this study to consider the effects of the diet upon the chemistry of a diseased stomach *per se*, rather than upon the ulcer when and if present. In this study no light is thrown upon the pathological changes that an ulcer undergoes during medical treatment. Only the immediate effects of the dietary restriction are studied. The observations extended as a rule over from two to five weeks in each case; the remote effects of medical treatment are not considered.

Procedure.—The routine examination of these patients upon admission consisted of a careful history and physical examination, extraction of fasting gastric contents, Ewald test breakfast and other chemical tests, stool examinations for occult blood and finally a thorough radiographic examination. A fractional test meal to establish the type of curve was also performed. After the collection of all data, the patient was placed upon a restricted diet for a period of from two to four or more weeks. During the period of observation, fractional test meals were taken every few days. The type of the acid curve, its maximum height, its duration, the presence of hypersecretion, the emptying time, etc., were carefully noted for any changes that could be interpreted as due to the form of treatment being instituted. Clinical notes were made from time to time so as to ascertain a possible

parallel between improvement in subjective symptoms and a change for the better in the disturbed chemism of the organ. The fractional test meal was chosen as the best laboratory method for the demonstration of the phases of gastric digestion. By this method, so intimately associated with the name of Rehfuess, the height and duration of the acid secretion can be computed and plotted, evidence of pathological hypersecretion becomes manifest and the emptying time of the organ accurately estimated for a given and constant test meal (oatmeal gruel in this study).²

BASIC PRINCIPLES OF SO-CALLED ULCER DIETS.

There are certain general characteristics which are common to all forms of so-called ulcer diet. These are:

1. Administration of highly nutritious foods in small quantities, frequently repeated.
2. Neutralization of the free acid of the digesting stomach.

This is accomplished by administering high proteid foods, mainly milk, with the addition of eggs and later of cereals. Antacid salts are commonly employed to assist the neutralizing action of the foods.

An improvement in the motility of the stomach is sought in cases where emptying time is delayed. In the series studied, patients were subjected either to a Lenhartz diet, carried out without modification, or to a diet similar to the one advocated by Sippy.³ In the diet as we administered it, milk and cream in small doses (2-3 oz.) were given hourly during the day for the first three days; cereals were then added twice daily. At the end of a week, two eggs a day were added and puréed vegetables were gradually introduced. All forms of meat were withheld until the third week. Antacids in small quantities were given between feedings; bismuth was a constant component of these powders. Occasionally, gastric lavage for food stasis was employed, though this was fortunately rarely indicated.

Before proceeding to discuss the general effect of such a diet upon the gastric chemism, let us study the individual chemical reaction which results upon the introduction into the stomach of these various foods. As a control one should keep in mind the normal curve of secretion, while employing oatmeal gruel as a test-meal or the usual Ewald test breakfast. Take, for example, the reaction of a patient with a normal curve to a test-

meal consisting of 240 c.c. of milk. One notes immediately the fact that milk causes a high total acidity (up to 128 c.c. N/10 sodium hydrate per 100 c.c.) ; that the height of the acidity is maintained for 3 to 4 hours ; that free acid is present in larger quantities than usually met with in cereal or bread test breakfasts ; and that the stomach takes considerably longer to empty itself. The acid-binding qualities of milk are apparent in only the first half hour ; thereafter to the end of digestion free acid is liberally present.

Compare this with the exaggerated effects of a combination of milk and eggs. The total acid curve mounts still higher, reaching a maximum of 146 per cent. ; free acid becomes evident after one and one-half hours, and is present in strong concentration during the remainder of the digesting cycle. Emptying time is as a result prolonged, even allowing for the large amount of test meal given. A free hypersecretion of concentrated acid persists after the disappearance of the last traces of food.

The behavior of a cupful (280 c.c.) of clear bouillon as recommended in the von Leube diet is similarly interesting. No considerable binding of acid is at any time demonstrable. A high acidity, both free and total, quickly takes place and is maintained for two hours ; thereafter the stomach is empty.

The conclusions to be drawn are: Milk is a strong acid stimulant, and is only slowly emptied through the pylorus. The combination of milk and egg is a powerful stimulant to gastric secretion causing a hyperacidity and hypersecretion with prolongation of the emptying time. Bouillon fails to bind acid and is a milk digestive stimulant.

The justification for the frequent repetition of food in small quantities is seen by observing the effect upon the secreting organ of three ounces of milk repeated hourly. While the total acidity remains moderately high (average 80 per cent.), very little free acid is allowed to accumulate. As the free acid curve tends to rise toward the conclusion of each 15-minute period, it meets the new portion of milk imbibed and is immediately neutralized. Since peptolytic digestion occurs only in the presence of free acid, it is apparent that insofar as actual splitting of the compound proteid molecule is concerned, this stomach is really inactive.

If to this hourly administration of milk we add an alternating small dose of antacid salts (bismuth and bicarbonate, or magnesia and bicarbonate in 5-grain doses of each as advocated by Sippy,³ we are enabled to note (Chart 5) that free acid entirely disappears and that even the total acidity is strongly controlled averaging only 43 per cent. Peptolysis is absent throughout.

If the reduction of total acidity and the complete neutralization or combining of free acid were the aims of this form of medical treatment then surely this plan of alternate milk and antacids fills all the requirements, and offers the stomach the optimum conditions of anacidity for the cure of an ulcer.

However, of late much doubt has been thrown upon the hypothesis that ulcer of the stomach is due to the corrosive action of gastric juice. This theory really gained headway before the realization by the older clinicians of the fact that duodenal ulcer outnumbers in frequency gastric ulcers. The charted curve of acidity in the duodenum during digestion regularly fails to show the presence of any free acid, nor is the total acidity at any time high. Our approved methods of medical treatment aim to accomplish in the stomach, the very conditions of combined or neutralized acidity which are regularly and normally present in the so-called juxta-pyloric portion of the stomach or duodenum. Yet under these very highly desirable and optimum conditions of anacidity, duodenal ulcers appear and grow.

A closer study of the entire subject tends only to raise more doubts as to the *modus operandi* of cure of ulcers by medical treatment. The causal relationship between the hydrochloric acid and ulcer has not been proven, nor are we able unquestionably to accept the hypothesis that the cure of ulcer depends on the neutralization of the free acid of the stomach.

However, clinically, the beneficial results of medical treatment cannot be denied or gainsaid; it behooves us, therefore, to proceed to study the effects of days and weeks of the administration of such a regimen on the gastric secretion.

EFFECT OF MEDICAL TREATMENT ON GASTRIC ACIDITY.

Of the 34 cases which constitute the basis of this study, 11 cases had an iso-secretory or hypo-secretory curve, and 24 a hyper-secretory curve of acidity. (Reh fuss,⁴) Let us assume

that a beneficial effect of the medical treatment would be the diminution of an excessive hyperchlorhydria and a return to a normal or hypo-secretory type of curve. In the series observed, medical treatment succeeded in causing a definite lowering of the acid curve in six of the 11 cases with iso or hypo-secretory curves; five of the 11 were unaffected or unchanged. Of the cases with marked hyperacidity, numbering 23 in all, only seven showed a lowering of the acid curve; in the remaining 16 the curve remained high in spite of persistent and rigid dieting and rest in bed.

A vital question must occur to one's mind at this point. Is the reduction of hyperacidity which takes place during medical dietary treatment paralleled by clinical improvement, and conversely is a resistant hyperacid curve accompanied by persistent gastric symptoms?

Taking the cases as a whole, 13 were chemically improved, 21 were not, a net chemical improvement of only 38.3 per cent. Yet *clinically* 25 of the 34 cases were discharged from the hospital free of symptoms, a percentage of 73.5 per cent., not quite as high as those presented by Lenhartz or von Leube but still high. Of the 13 favorable cases in which the acid curve was reduced and the hyperacidity obliterated, 12 left the hospital symptom-free.

On the other hand, of the 21 cases with a *persistent* high acid curve in spite of treatment, 13 or 62 per cent. were markedly *clinically* improved. The remaining 38 per cent. (8 cases) left the hospital with the same curve as on admission and clinically unimproved by their treatment.

A certain parallelism between chemical and clinical results is obvious.

TABLE I.

	Number of cases.	Clinically improved.	Clinically not improved.
Acid curve, reduced.....	13	12 or 92%	1
High acid curve, persistent....	21	<u>13 or 62%</u>	8 or 38%

The underlined group is the interesting group, namely those in which the curve remained acid-fast and yet the patients left the hospital free of symptoms.

EFFECTS OF RESTRICTED DIET AND REST IN BED UPON GASTRIC HYPERSECRETION.

The fractional test meal is an ideal medium for the demonstration of hypersecretion. As the successive samples are removed from the stomach at quarter-hourly intervals, the proportion of food or cereal to the digestive fluid becomes smaller. With normal motility the last portion of the gruel disappears at two hours. It is at this time that hypersecretion when present becomes evident, for from this point, one removes every 15 minutes a sample of secretory fluid unadulterated by food. This is the pure secretion of gastric tubules; it is usually profuse so that large amounts can be removed, and it continues sometimes for several periods or even hours after the food has passed the pylorus. In exaggerated instances as much as 30 c.c. of pure acid fluid can be removed every five minutes.

The exact clinical significance of alimentary hypersecretion (we shall omit from present discussion continuous hypersecretion) has yet to be determined. Strauss,⁵ Zweig,⁶ and others who noted the phenomenon, described it as a pathological factor and ascribed its presence to the existence of an organic lesion of some part of the alimentary tract. Gastric or duodenal ulcer, chronic appendicitis or reflex irritation of gall-stones were and are all held responsible for the stimulation of this excessive fluid. But opinion is not uniform on the significance of its presence, and we need only quote Rehfuß,⁷ who in a recent article spoke of hypersecretion as being of no pathological importance and of its presence in several normal controls.

The period in the cycle of digestion in which hypersecretion is most evident is the period following the evacuation of the food. The titer of this hypersecretory fluid is usually high in acid, averaging from 60 to 80 per cent.; practically all of it is free acid, only a very small percentage is present as combined acid and practically none as acid salts. It is at this period that the pains of ulcer are often most marked; this remains true whether we ascribe the pains to the direct action of the excess of free acid upon the ulcer or the stomach wall, or whether we attribute the pains to the exaggerated peristaltic waves or hunger contractions which become evident during this part of digestive cycle.

Whatever the significance, hypersecretion is a common phe-

nomenon in gastric and duodenal ulcer; it was present twenty times in this series of 34 cases, appearing toward the end of the period of food digestion as a post-digestive or alimentary hypersecretion.

We are interested in knowing what effect, if any, medical treatment consisting of a marked restriction in food and rest in bed has upon this factor. A close association is noted between hypersecretion and hyperacidity; 16 of the 20 instances of hypersecretion were in cases with gastric hyperacidity. In 9 of the 20 cases where present, hypersecretion was markedly improved or caused to disappear by medical treatment, a percentage of 45 per cent. In the other 11 cases, or 55 per cent., no effect was noted. The beneficial results of treatment were often very marked in this respect.

Many well marked examples of the effect of medical treatment in causing the disappearance of hypersecretion could be demonstrated in the cases of this series. In fact, this is one of the most striking effects of medical treatment when a good result is present.

Much disappointment was felt in that the beneficial effect was not accomplished in a greater percentage of the cases. Eleven cases or 55 per cent. were in no wise modified by either the restricted diet, the use of antacids or the physical and psychical rest accompanying the stay in the hospital. It is hardly possible to explain the failure in this not inconsiderable percentage of the cases.

Does improvement in hypersecretion accompany a reduction in hyperacidity, or, in other words, are both factors affected in the same manner by the medical treatment? A reference to Table II is instructive.

TABLE II.

Hyperacidity reduced, hypersecretion improved, clinical result good, 6 cases.

Hyperacidity reduced, hypersecretion improved, clinical result poor, 1 case.

Hyperacidity *not* reduced, hypersecretion *not* improved, clinical result good, 8 cases.

Hyperacidity *not* reduced, hypersecretion *not* improved, clinical result poor, 3 cases.

Hyperacidity *not* reduced, hypersecretion improved, clinical result good 2 cases.

Total, 20 cases.

It is thus seen that in all but two cases, the last two in the table, both factors were identically influenced, either both being beneficially influenced or neither one affected for the better. The frequent co-existence of these two factors, and their similarity of behavior under treatment, suggests a close association between these two factors.

As for the clinical results of treatment, the same inconsistency is naturally seen in this group as in those in which only the effect on hyperacidity was considered. While in 11 cases, or 55 per cent., the treatment failed to affect the hypersecretion, yet 16 of the 20 cases, or 80 per cent., were discharged from the hospital clinically much improved and actually free of symptoms.

EFFECT OF MEDICAL TREATMENT ON GASTRIC MOTILITY.

What is the result of our medical efforts upon delayed emptying of the stomach and persistent food residue?

Before proceeding directly to a critical review of the cases, let us consider for a moment as a preliminary the question, "Is the fractional test meal a good medium by which to judge the motility of the stomach?" There has been much discussion as to the relative merits of the roentgenological method for demonstrating delayed motility and on the other hand of chemical methods for demonstrating postponed emptying. Both methods have undoubted value, and either method in the hands of a competent technician and observer is beyond question a reliable index of motility.

The fractional test meal by allowing observations at such short intervals as one-fourth hour, seems to have the advantage over both other methods, these latter being limited to a single observation at the termination of a stated interval. With the fractional method one can state within a few minutes just when the last remnant of food has passed the pylorus. In every instance where a six-hour residue was reported by X-ray methods, a chemical delayed motility was also evident with the Rehfuß method. On the other hand, innumerable instances are met with in which slighter grades of delayed emptying are evident by this chemical method, which evade entirely the roentgenologist.

Using 500 c.c. of thick oatmeal gruel as a test breakfast, normal motility with the fractional method may be regarded as one and three-fourths to two hours; in cases with moderate degrees of atony up to two and one-fourth hours.

In this series of 34 cases of ulcer or ulcer-suspects, there were 13 instances of delayed motility using the fraction test as a criterion. The time of emptying varied from two and one-fourth hours to a maximum time of three and one half to four hours. It is in this field that the optimum effect of medical treatment was obvious.

In all but two of the cases of the series emptying time was definitely, in many of them markedly shortened, a proportion of 85.4 per cent. Still more striking is the fact that clinical improvement as noted ran quite parallel with the reported improvement in motility. All of the eleven cases in which improved motility was accomplished were discharged free of symptoms, while the two cases in which the delay in motility resisted our efforts were discharged unrelieved of subjective symptoms.

A reduction of acidity did not always accompany an improvement in the motility. Thus of the 11 cases with improved emptying time, in only three was hyperacidity reduced, while in the remaining five the high acid curve persisted. In spite of the persistent acidity all these cases were discharged free of symptoms.

Hypersecretion was noted six times in association with delayed emptying. In four of these six instances it was not relieved by the treatment; in these cases, though the hypersecretion persisted, the pyloric stenosis or pylorospasm was relieved and the patient was discharged free of symptoms.

The following is a tabulation of the results of motility test which followed treatment and the day upon which the test was taken.

	Motility before treatment.	Motility after treatment.
Case 12	2¼ hours	1¼ hours 12th day
Case 16	3 hours	2¼ hours 6th day
Case 17	2¾ hours	*2¾ hours 12th day
Case 21	2¼ hours	*3 hours 9th day
		2¾ hours 15th day
Case 23	3½ hours	2¾ hours 7th day
		1½ hours 10th day
		1¾ hours 12th day
Case 24	3 hours	2½ hours 3rd day
		2½ hours 8th day
Case 27	2½ hours	2¼ hours 6th day
Case 28	2½ hours	*2¾ hours 3rd day
		2¾ hours 12th day
Case 30	2½ hours	1¾ hours 10th day
Case 34	4 hours	1½ hours 26th day

*No improvement.

Discussion.—Having studied in detail the effect of restricted diet and rest in bed upon each of three factors which show deviation from the normal, let us try to evaluate the relative importance of the change which takes place in each of them. Let us attempt to estimate the relationship between clinical relief from symptoms (cure?) and physiological or chemical benefit.

Does medical treatment reduce the hyperacidity of ulcer, and if so, is this relief responsible for the clinical improvement?

Does restricted diet cause the disappearance of hypersecretion, and if it does, is this the factor that relieves the symptoms?

Finally, can delayed motility in the presence of ulcer be relieved by the feeding of small quantities of food, often repeated? And if so, is this the factor that brings about apparent cure of symptoms? We will discuss these factors individually and collectively recapitulating the findings in the body of the paper.

Medical treatment resulted in a net reduction of acidity in 13 out of a series of 34 cases (38 per cent.). If we wish to consider only those cases with definite hyperchlorhydria, omitting all cases with iso- or hyposecretory curves, the percentage of net lowering of acidity is still lower (30.3 per cent.). Of these 13 cases chemically benefited, 12, or 92 per cent., were discharged free of symptoms. Loose reasoning might lead one to see a relationship of cause and effect in these figures. But if we analyze those cases in which treatment failed, to invoke chemical relief and these comprise the larger proportion, we find despite this fact, clinical relief in 13, or 62 per cent., of them. In other words, of 25 cases discharged from the hospital free of symptoms and apparently well, 13, over one-half, retained the same height and type of acidity as on admission. If clinical cure depended upon relief from hyperacidity, then the optimum result should be in this class of case. Almost 80 per cent. of the cases with hyperchlorhydria left the hospital clinically relieved, yet only 30.3 per cent. of these showed chemical relief from the excessive acid production.

We can draw two inferences from these figures. First, only a small percentage of ulcer cases react to medical treatment by showing a reduction of acid produced during digestion (38 per cent.). Second, clinical improvement can take place independently of whether the hyperacidity is relieved or whether the case remains acid-fast.

As regards *hypersecretion*, a similar conclusion is soon arrived at. Twenty cases in the series showed hypersecretion; in nine cases, or 45 per cent., medical treatment caused the disappearance of this abnormal factor, in the other 11, or 55 per cent., of the cases, medical treatment failed to alter the hypersecretion. Yet 80 per cent. of these cases left the hospital apparently well. Exactly 50 per cent. of the clinically cured cases still retained hypersecretion upon discharge from the hospital, and yet subjectively the pyrosis, heartburn, and pain had disappeared.

From these figures, we can again draw two conclusions. Medical treatment consisting of restricted diet and rest in bed causes the cessation of hypersecretion in 45 per cent. of the cases, a fair proportion. Second, clinical improvement takes place as often in cases with persistent hypersecretion as in those relieved of their excessive flow of gastric juice, and is apparently not dependent upon it.

Finally, we shall discuss the relationship between improved gastric motility and clinical relief. There were 13 cases of delayed motility; in 11, or 85.4 per cent., medical treatment succeeded in alleviating this symptom and reducing the emptying time to normal. All of these 11 cases were in addition freed of subjective complaints. Two cases resisted treatment (motility unimproved) and they were discharged clinically unrelieved.

There are no instances of clinical cure that could be solely attributed to reduced acid secretion when delayed motility was present as a complicating factor. Unless relief is had from the pylorospasm, we fail to note the disappearance of subjective symptoms. By stating that relief of pylorospasm results in clinical cure and is probably the cause for amelioration of symptoms, we have only answered part of the question. For most of the cases in this series were not complicated by delayed motility; they were cases with hyperacidity and hypersecretion but normal motility. We have tended throughout this study to discredit the idea that clinical relief is built upon a reduction of the hyperacidity or hypersecretion, and can and does take place independent of chemical change.

Then, to what change in the physiology or pathology of the stomach can one attribute the relief from symptoms in cases not complicated by pylorospasm? No data in this study throws any light on this question. The answer must be sought and will prob-

ably be found in the radiographic studies of Ginsberg, Tumpausky and Hamburger,⁸ Carlson,⁹ Hardt,¹⁰ and others.

These authors have demonstrated the close relationship between subjective pain and exaggerated peristalsis or hunger contractions. It is quite likely that restricted diet and bed rest have a quieting influence upon gastric contractions, diminishing the amplitude and violence of these contractions. Reduction of acidity and secretion may or may not accompany the reduced tonus of the stomach.

This study has demonstrated that in fair proportion of the cases medical treatment caused reduction of hyperacidity and of hypersecretion. To what can be attributed the failure to effect this change in a far larger percentage? The answer probably lies in the fact that the marked restriction of food intake is not maintained for a long enough period. Almost all schedules of "ulcer diet" increase the number and amounts of food rapidly, particularly during the second week. In fact, Lenhartz already introduces meat on the 10th day. It is a common observation that hospital patients complain bitterly of being starved in the early days of dieting, yet it is just at this period that they report the disappearance of their annoying subjective symptoms. The end of the second week which usually marks the period of increased alimentation is a common date for reappearance of symptoms of pain and heartburn. It has further been shown that it takes up to the sixth to the 10th day for the acid secretion to show beneficial result as evidenced in a lowering of the curve. The earliest effect of the marked food restriction is probably diminished tonus and contractions and increased rest to the stomach walls. The later effect is a fall of acidity and secretion. The full beneficial result, particularly on the hydrochloric acid production, is probably defeated by a too early return to a more liberal diet. The introduction of meat during the second or third week is harmful; meat-free diet should be continued for weeks or even months.

It is probable, too, that a diet based in its earlier stages on milk alone, rather than on milk and eggs as in the Lenhartz diet, is to be preferred. The amount of secretion called forth in the digestion of a milk-egg diet is far greater, and the emptying time is much longer for a milk-egg mixture than for a milk meal, or certainly for a cereal meal. Personally, I would favor rather a

greater liberality and earlier introduction of cereals, and more restraint in the use of eggs.

As has been demonstrated in a previous paper by the author,¹¹ antacids should be used often and in small doses lest they in themselves increase or stimulate the acid cells to production. Nor can the influence of the psychical factor be overlooked. Mental contentment during a course of treatment is essential. A noisy hospital ward, the bad example of a bed-neighbor who is a neurotic and constantly complaining of the food, the treatment and his symptoms; mental disturbance over economic conditions at home, are all influences that tend to cause persistence of secretory and motor abnormalities and deprive the patient of the full benefit of his rest in bed and dietary cure. This principle applies as well to the ulcer cases as it more evidently does to the cases of functional secretory disturbances or neurosis. A patient who is unhappy during his treatment is unlikely to get well.

One cannot overlook the physical difficulties of treating many cases by "ulcer diet" in a busy hospital ward. The war intensified these difficulties many times by creating a shortage of nurses. It is not an easy matter for a limited number of ward nurses to serve milk every hour, give antacid medication on the alternating half hours and look after the general wants of the patients in addition. Cereal which has been delayed in transit and becomes cold is tasteless and to many patients disagreeable to eat. I fear that many of the failures to obtain better results are due to the physical inability of even the best hospital staff to live up to the details and the schedule of these time and attention-consuming dietary regimens.

Segregation of cases and special nursing facilities would solve many of these difficulties. This probably explains why the results in private practice are far better and more permanent than those in the hospital public service.

Cases that fail to show clinical and chemical improvement are of two kinds, those with advanced indurated ulcers which are resistant to treatment and those with marked neurosis. The first class can be reduced in number by more persistent and more protracted treatment. The second group requires the expert care of a neurologist, often of a psychoanalysis. Mistakes in diagnosis undoubtedly form a third group of not considerably proportion. The beneficial effects of medical treatment are seen in those cases

of hyperacidity, hypersecretion and pylorospasm accompanying latent cholelithiasis or chronic appendicitis, as well as in ulcer cases or functional secretory disturbances. This remark applies to immediate results rather than to permanent benefits.

It is to be regretted that the cases in this series could not be observed for a sufficiently long period to determine the durability of the result, and to determine whether a relation existed between improved physiological function and permanent cure or relief.

There is a large field for research still open along these lines. Much remains to be learned regarding the physiology and pathology of the stomach during and after medical treatment. It is only by close study and observation that we will learn to account for our failures and learn to recognize the factors that contribute to the clinical cure of these patients.

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DISCUSSIONS.

DR. EINHORN: Dr. Crohn made the remark that it is difficult to explain why a duodenal ulcer should exist, because the duodenal contents are always neutral. Now, I would say that duodenal ulcers usually are situated near the pylorus, right nearby, just on the other side. The chyme must enter the duodenum. It cannot become neutral or alkaline just in a second, you know. It takes a certain amount of time, and it happens very quickly; but on the other side through the pylorus it touches and irritates that part, and that is the reason why ulcers are found there. But I found exceptions to these rules, too. I really have found instances in which the duodenal contents were acid; not only that means combined acids, but free acids were present in a case of ulcer where I demonstrated distinctly with the duodenal tube that we were in the duodenum, and still we have free hydro-

chloric acid right there at certain times in a patient with ulcer. I do not recollect in that particular patient whether the ulcer was situated in the duodenum or stomach; but I know exactly the case where that happened. The question why an ulcer can exist there is easily explained, because usually it is right near the pylorus, and over there the acids pour away, and it is a very favorable spot for an ulcer. Now, with regard to the methods of treatment in ulcer, and with regard to influencing the patients and the acid: I must say from my own experience with regard not only to ulcer, but to other conditions, too, that if we find too much acid or too little acid in a patient that while we improve the symptoms, in most of the cases there are exceptions to these rules; but in most of them, if we examine a patient a little later, we usually find the same type of acidity. I must say that, and every clinician I think knows of that. Now, with regard to the explanation why a patient should feel better after a treatment of an ulcer when he has a return to the type of acidity, just as before, as Dr. Crohn says, that indicates that the acidity alone is not the most important part of the treatment. It is the rest. I should think they should have a chance to recuperate. The resistance of the mucosa becomes better; the circulation becomes better established and in this way they feel better and better, notwithstanding a greater acidity; and clinically it is not really of such great importance. I mean that we should see that the patient should have no acidity. What we are after is to bring a patient back to his normal health.

DR. FRANKLIN W. WHITE, Boston: We all realize that the diagnosis of pancreatic disease is a difficult field. To say a word about the laboratory end of it, it seems to me easier to deal with duodenal contents obtained through a tube than with stools. Quantitative estimation of ferments is difficult; we have no really accurate method. Trypsin is the most useful ferment in diagnosis and only *marked* changes in amount have clinical value. We may find the ferments well preserved in the presence of marked pancreatic disease. This is unfortunate for diagnosis, but fortunate for the patient. The healthy part of the pancreas is doing its best to compensate for the damaged part and to produce a good, normal secretion. In the end, I think that our diagnosis must be largely clinical—helped out, to a certain extent by laboratory tests.

DR. ALVAREZ: It is refreshing to hear this paper of Dr. Crohn. Years ago I thought I knew a good deal about the origin of gastric ulcer, but the more I read the less I feel sure of. Accurate studies like these of Dr. Crohn often fail to give us the positive information which we desire, but they are very helpful in proving that the things we had accepted are false; and that we must make a new start in our search for truth. We do not know why an ulcer gives pain or why the pain goes away on giving food. In the textbooks you will find many conflicts of opinion as to the rationale of ulcer treatment. My

own conviction, after many years of seeing these ulcer patients returning with relapses is that our medical treatments do little good except perhaps in rare cases. In some cases the permanence of a cure may be due to the fact that the patient did not have an ulcer. Although many of my cases are not entirely cured by the surgeon, I believe his results are so much better than mine that I generally send him these cases as soon as I make the diagnosis. I treat only those who are in their first or second attack and who have the money and time to waste on a temporary cure. I treat them also when the diagnosis is not certain or when for business reasons they have to defer operation for a while.

DR. JONES: It seems to me that in the treatment of ulcer medically we should take the point of view of treating the patient that has the disease rather than the disease that has the patient. Personally, I have never been able to get satisfactory results from Lenhart's treatment. The Von Leube treatment and especially the Einhorn duodenal tube treatment, have been, in my hands, the most successful of any medical treatment. I must say that I am very much surprised at the remarks of one of the speakers with reference to the medical treatment of ulcer. A considerable number of surgeons with whom I am associated customarily refer patients with duodenal ulcer and with ulcer of the stomach to the medical side of the hospital for treatment, and if those cases are followed, if they are not lost sight of, when they leave the hospital, if we follow them up carefully with dietary treatment for a year or possibly two years after hospital treatment, I think we will find that they are not only symptomatically cured, but they are well, because if the patient is managed both from dietary point of view and to relieve those exciting causes of ulcer, which have nothing to do, so far as I can see, directly with ulcer of the stomach, but have to do through the manner of living, the daily life, the exercise and the things which irritate the patient through his nervous system and bring him into a state that makes him susceptible to ulcer, the real source of the trouble will be reached. Another point that comes up frequently in my clinical practice is the patient who has been operated on for duodenal ulcer and who has a recurrence of the ulcer, and I have quite a list of these patients that might be reported, they have been relieved by the Einhorn duodenal tube treatment from four to six weeks and the careful regulation of life and diet, but not for less than one year, and usually two.

DR. FRIEDENWALD: I should like to add a word regarding the cure of peptic ulcer: I do believe that ulcers can be cured medically in many instances, and this fact may be proven by means of X-ray examinations. Some years ago, Dr. Baetjer and I brought out in our X-ray studies of ulcer, that when a patient is given a rest cure treatment all symptoms gradually disappear, and the patient becomes comparatively well. This usually takes place in from 4 to 5 weeks. At the end of this time, however, if a second bismuth examination is

made, we often find the same characteristic signs as in the first instance, though the patient shows no symptoms whatever. In a series of ulcer cases that has been examined in from 3 to 4 weeks after an absence of symptoms, we have frequently found but little change in this defect or motility of the stomach. When these patients are given the ordinary diet their symptoms may recur in a short time. If treatment is continued, however, our experience has demonstrated that as the ulcer continues to heal the motility of the stomach returns to a more normal condition, and by making repeated X-ray observations over a long period of time we are enabled to determine not only the progress of healing, but also to observe when the ulcer has healed.

DR. REHFUSS: Surgeons say we have no right to call an ulcer one that is not shown on the operating table. I have seen many cases that were called medical ulcers. I found that 38 per cent. of the hundred cases had high acidity, but forty per cent. of our normal cases gave the same acidity, indicating that there probably could be no real association of acidity to ulcer formation, although it might aggravate ulcers. Certain cases of acidity, and others that went into the interdigestive phase where a hypersecretion was relieved, the return to the symptoms was accompanied by this clinical relief. Meat, the yolk of eggs, milk, produce a high acidity. The cereals, the fruits, and the green vegetables produce a low acidity, and you can work it out, and follow it out. I say we have got over a thousand curves. There is no doubt about it at all, and therefore it would seem that the best form of diet would be the one to promote low acidity. Therefore, the question resolves itself into something more than a question of acidity.

I have these cases X-rayed before and after to see whether there is any change in the X-ray picture of the ulcer condition, or in its motor functions. We can measure with a tube more accurately than the X-ray man can. At least, I have taken the stomach of the individual after it has been pronounced empty on the fluoroscopic screen and still got bismuth out without any difficulty whatsoever, although I really admit that the amount of bismuth is practically negligible. Now, I believe that these cases do get well. I believe that a certain number of ulcers are acute ulcers. I make this statement, that I see as many cases of post-operative gastric ulcer as I see gastric ulcer; and that statement I can substantiate by looking up my books. These cases are coming in, showing that the operative thing is not settled. If you have not studied the normal gastric physiology, how can you cut with a knife the part that you do not even know anything about?

DR. CROHN (closing): The difference between the various diets is not important. They all act about the same.

It is not so much the pyloric spasm, but the retention of the acid; when the case is beneficially influenced by diet, improvement of the motility takes place; the tonus of the stomach, the balloon tonus, is diminished; the constant tonus of the stomach is relaxed; peristalsis

is less active; the pylorus opens and functionates more normally; that is probably the more likely explanation why ulcers heal, rather than attributing it to the diminution of acid. Regarding the percentage of cures in medical treatment, Lenhartz reported something like 85 per cent., and he was a strong competitor of Van Leube who said he cured about 87 per cent. We know now that probably they did not cure their cases; there are no statistics on dietal end results. There is no doubt in my mind that ulcers can be healed medically, and if they are not of long standing, I am sure that you can get good results with them. Dr. Friedenwald said he had seen the cure of an ulcer radiographically demonstrated years ago.

RESULTS OF DUODENAL ALIMENTATION IN PEPTIC ULCERS.

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NEW YORK.

It is just nine years since I had the pleasure of reporting to you on duodenal alimentation. While this mode of treatment is applicable to a variety of conditions, its main field is peptic ulcer. It was in this disease that I first employed it. A number of later papers, likewise, refer to the same condition.

Numerous clinicians here and abroad have administered duodenal alimentation in gastric and duodenal ulcerations with benefit. Among these I mention S. Basch, A. F. Chace, R. F. Chase, H. Fischer, J. A. Friedenwald, M. Gross, I. W. Held, W. V. Hayes, J. C. Johnson, A. A. Jones, Clement R. Jones, J. A. Lichty, George L. Laporte, Paul Lazarus, Willy Meyer, C. J. MacGuire, William Gerry Morgan, L. Montané, J. A. McCafferty, H. A. Rafsky, Charles G. Stockton, F. Pages, N. de Rosas, L. Stieglitz, Franklin White, J. G. Wells, J. R. Verbrycke, and many others.

It appeared to me of interest to discuss today my experience with regard to the results of duodenal alimentation in peptic ulcers.

Peptic ulcers may be divided into: (1) Uncomplicated cases of moderate duration; (2) uncomplicated cases of very long duration; (3) cases complicated by recurrent hemorrhages; (4) cases complicated by obstruction of the pylorus; (5) cases complicated by perigastric adhesions or perigastritis.

All uncomplicated cases (Groups 1 and 2) are amenable to medical treatment, especially to duodenal alimentation. Group 3 should be given a trial by duodenal alimentation. If hemorrhage returns pretty soon after this mode of treatment an interval operation (gastro-enterostomy, sometimes, with pyloric occlusion) should be performed. Groups 4 and 5 usually require surgical measures. However, if the stenosis of the pylorus has not reached a high degree, medical treatment can first be tried, and if neces-

sary, be followed by stretching of the pylorus through the internal route. In perigastritis the rest period during duodenal alimentation may bring sufficient relief to forestall an operation. The same applies especially to old people or greatly weakened individuals, who could not well stand the hardships of an operation.

Groups 3 and 5, and 4 when the pyloric stenosis is still permeable to the duodenal tube, may when they are in a weakened condition first advantageously be subjected to duodenal alimentation, in order to be brought into a more fit condition for a future operation.

The results of duodenal alimentation in peptic ulcer will depend to a great extent first upon the proper selection of cases and upon a correct diagnosis, second upon a conscientious and detailed handling of this mode of treatment.

Careful observation of the patient, especially in a hospital with systematic examinations for a few days, will usually suffice to make a well-balanced diagnosis. The details of duodenal alimentation can be easily acquired after a short period of study and practice, so that every practitioner could carry it out.

In order to study the results of duodenal alimentation in peptic ulcer I select my material among my private patients of the last nine years. Most of these I could follow for quite a while after treatment, some of them almost up to the present time. To be sure there are some among them in whom the definite result is unknown to me. Selecting, however, those I do know as a basis, the others will probably show approximately similar results, so that the statistics of my material will not be far from the truth.

All in all 315 cases of peptic ulcer were treated by duodenal alimentation. Among these there were 247 with definite localization of the ulcer, as follows:

LOCALIZATION OF THE ULCER IN 247 PATIENTS.

	Cardia (35-42.)	Lesser Curva- ture (43-53).	Pylorus (54-55).	Duodenum (Below 55).
Males.....	10	43	36	65
Females...	14	38	19	22

The immediate results were satisfactory in all excepting four. That means subjectively the patients felt better, could stand more

food and a greater variety without suffering, usually could follow their vocations a few weeks after treatment, and had a gain in weight varying from 10 to 20 pounds. The string test, as I have observed in a former paper, does not always become negative in the usual two weeks' duodenal alimentation given the patient. In about half the cases the string test remains positive at first, to disappear some weeks later on. In others it remains positive even a longer time after treatment.

It would be perfectly legitimate in these cases to try and extend the period of rest accorded the stomach for a longer period of time. The patients, however, notwithstanding the positive string test, are usually feeling so well that I do not consider it right to subject them to worry and treatment. It is different in cases with manifest complaints. Here another period of duodenal alimentation should be given right away without question.

In looking over the entire list of 315 patients I find 31 which require comment. I give them in a table.

Among these, four had the alimentation without benefit; eight required operation soon after the period of duodenal feeding; four for pyloric obstruction, three for recurrent hemorrhages, and one for perigastric adhesions. Four had to be operated upon later on (*i. e.*, 1 to 5 years subsequent to the duodenal alimentation treatment), three of these for pyloric stenosis, and one for recurrent hemorrhages. In 15 there was a recurrence of ulcer symptoms 2 to 5 years after the first duodenal alimentation treatment, necessitating a new period of feeding with benefit to the patient.

There were no deaths encountered during the duodenal alimentation. The procedure of duodenal alimentation is certainly not connected with any risks to life.

To be sure there are a great many parents among the 315, whose results are not known to me any more, for they cover a period of nine years.

I though it might be of interest to look over the results of more recent years, as they are better known to me. I, therefore, take 1915, 1916, and 1917 as examples.

In 1915, 47 patients were treated with duodenal alimentation. The results were good, excepting one not improved, one patient with duodenal ulcer and great benefit from treatment (gained 30 pounds in weight) had developed cancer of the lesser curvature two years later, from which he died subsequently. One

patient required operation for pyloric stenosis. Two required another course of treatment three years later (1918).

I.—TABLE OF CASES OF PEPTIC ULCER FROM THE 315
TREATED BY DUODENAL ALIMENTATION, IN WHICH
IMPERFECT RESULTS HAVE BEEN OBTAINED.
(1910-1918).

Name.	Date.	Location of Ulcer.	Remarks.
Miss Elsa S.	10-7-10	Lesser curvature.	Recurrence about 1917.
H. T.	10-27-10	Pylorus.	Recurrence about 1916.
Richard S.	1-6-11	Duodenum.	Recurrence about 1916.
Odie S.	1-6-11	Duodenum.	Recurrence 1912 (9-18-12).
A. D. S.	2-16-12	Duodenum.	Recurring hemorrhage; operation.
Miss C. G.	11-24-12	Duodenum.	Improved; not cured. Recurrence, 9-16-14.
H. P. R.	4-25-12	Lesser curvature.	Operation four years later.
E. C. M.	4-6-12	Lesser curvature.	Recurring hemorrhages six months later; operation.
W. J. Mc.	5-28-12	Duodenum.	Duodenal alimentation previous to operation 5-24-12.
F. F. C.	9-19-12	Pyloric stenosis.	Recurrence 2-19-16.
M. P. D., Jr.	1-12-13	Pylorus.	Recurrence.
Mr. G.	4-12-13	Lesser curvature.	Two years later, hemorrhage; operation.
L. R.	1-4-14	Pylorus.	Recurrence 1916.
S. S.	2-16-14	Duodenum.	Postoperative dyspepsia; no benefit.
Dr. S. H.	4-5-14	Pylorus.	Hemorrhage pretty soon after duodenal alimentation; operation.
Miss M. G.	5-3-14	Lesser curvature.	Recurrence 1917.
Miss H. P.	9-13-14		Not improved.
Dr. J. E. C.	1-15-15	Duodenum.	Recurrence 9-11-18.
Dr. J. P.	4-14-15	Duodenum.	Recurrence 12-9-18.
John D.	4-30-15	Cardia.	Not much improved.
Miss F.	10-13-15		Required operation later.
M. A.	6-16-16	Duodenum.	Not improved.
Mrs. E. R.	8-31-16	Lesser curvature.	Recurrence two years later.
Mr. L.	11-25-16	Duodenum.	Operation later for pyloric stenosis.
F. J. N.	3-17	Pylorus.	Improved; later hemorrhage; therefore operation.
		Stenosis of pylorus.	
Jacob C.	3-17	Pylorus.	Required operation.
A. B. H.	4-15-17	Pylorus.	Recurrence 10-20-17.
Mr. S.	5-19-17	Duodenum.	Recurrence, operation.
Mr. S.	1-18-18	Prepyloric ulcer.	Required operation on account of perigastric adhesions.
Mrs. P.	2-20-18	Duodenum.	Hemorrhage; operation.
Dr. S.	4-18	Duodenum.	Required operation.

II.—TABLE OF CASES, TREATED BY DUODENAL ALIMENTATION DURING 1915-1917, SPECIFYING THE IMPERFECT RESULTS.

Year.	Number Treated.	Name.	Location of Ulcer.	Remarks.
1915	47	John D.	Duodenum.	Not improved. Relapse in 1918; duodenal alimentation with benefit. Relapse in 1918; duodenal alimentation with relief. Carcinoma vent. in 1917. Operation later.
		Dr. C.	Duodenum.	
		Dr. P.	Pylorus.	
		Mr. P. Miss F.	Duodenum. Duodenum.	
1916.	39	Mrs. R.	Lesser curvature.	Relapse in 1918. Later required operation for pyloric obstruction.
		Mrs. L.	Duodenum.	
1917.	46	J. C.	Pylorus.	Operation for pyloric stenosis pretty soon after duodenal alimentation.
		Mr. N.	Pyloric stenosis.	Operation for pyloric stenosis and hemorrhage 2-3 months after treatment.
		Mr. S.	Prepyloric ulcer.	Operation for pyloric stenosis 18 months after duodenal alimentation.

In 1916, 39 patients with peptic ulcers were treated by duodenal alimentation. One required operation for pyloric obstruction, another one had a return of ulcer symptoms in 1918.

In 1917, 46 ulcer patients were fed by the duodenum. Three cases required operations for pyloric obstructions. One (J. C.) pretty soon after the alimentation period; another (N.) about three months later; the third 18 months later.

As can be seen at a glance from the above figures the results of duodenal alimentation in peptic ulcers are very favorable. Not taking the late recurrences of ulcer symptoms after two years into consideration the good results would be 95 per cent. Including later recurrences and necessary operations after a two-year period of benefit as failures we would still have recoveries in 90 per cent. of those treated.

If one looks at the special varieties of peptic ulcers with regard to the results of treatment by duodenal alimentation one finds that the imperfect cases are principally encountered in complicated

ulcers of long standing and in pyloric and duodenal ulcers with or without recurring hemorrhages. Ulcers of the cardia and lesser curvature give the greatest percentage of cures.

It may be of interest to mention that we applied duodenal alimentation in six cases of abundant recurrent gastric hemorrhages of the severest type, immediately after this event and have achieved perfect cures. They are described in the subjoined table.

III.—TABLE OF CASES OF ABUNDANT AND REPEATED GASTRIC HEMORRHAGES, IN WHICH DUODENAL ALIMENTATION WAS ADMINISTERED DURING OR IMMEDIATELY AFTER THE HEMORRHAGE.

Name.	Date.	Remarks.
Mrs. McQ.	10-4-11	Frequent profuse hemorrhages, constant vomiting, had lost 50 pounds. Treated by duodenal alimentation, gained 60 pounds; remained well ever since.
Mrs. M. K.	10-14	Had lost two-thirds of her blood; recovered after duodenal alimentation.
L. S.	2-15	A patient with duodenal ulcer, and extensive hemorrhage; tube introduced after the bleeding had stopped.
Miss A. de P.	11-16	Had 3-4 extensive hemorrhages in three days, with syncope. Tube introduced during the bleeding period with marked benefit.
Mr. O.	4-19-18	Had several recurrent hemorrhages in one year; in the last hemorrhage he had lost more than half of his blood. Tube applied during the hemorrhage with benefit.
H. P.	4-21-18	Duodenal tube failed to enter the duodenum in 48 hours. Another tube with bead attachment was introduced and passed the pylorus in 18 hours.

Inasmuch as the results will more or less depend upon the handling of the cases it will not be superfluous to give a short outline of the entire plan of the treatment.

The diagnosis of peptic ulcer having been made and the possibility of malignancy having been excluded with great probability, the patency of the pylorus is then ascertained (retention meal of rice or rice and raisins and the duodenal bucket string test). The pylorus not being stenosed (or not strictured to a great extent) the duodenal tube is then introduced and feeding by the tube begun

as soon as the latter has reached the duodenum. The period of duodenal feeding is ordinarily two weeks, but can be extended for a longer time, whenever this appears appropriate. During the tube feeding period the patient receives daily eight meals in two-hour intervals, consisting of milk (7 ounces), one egg, and one to two tablespoonsful of lactose; besides one pint to a quart of saline twice daily, likewise through the tube. As a medication we usually give subnitrate of bismuth gr. xxx, in conjunction with calcined magnesia gr. ii-v t.i.d., in a wineglassful of water by the mouth. A mouth wash should be frequently applied, and the bowels should be made to move once daily either by medication or by enemas. For nervousness, Tr. of valerian or a bromide can be employed and it is best given in the feeding through the tube, and for pains codein or atropine administered in the same manner. Patient is ordinarily kept in bed during the first week of the tube feeding; the second week he is up gradually more and more every day. After the two weeks of tube feeding are completed, the tube is withdrawn and nourishment given by mouth, at first principally liquids, but very soon semi-solids, and in about three days also light solid foods. At first patient is fed every two hours, but as soon as he is on a liberal diet three ordinary meals and two small meals in between are instituted. The above-mentioned bismuth medication is continued for a month or two longer and then gradually discontinued.

In recommending duodenal alimentation for the treatment of peptic ulcers I like to lay emphasis on two important points this method enables us to carry out—namely, rest of the affected organ and adequate nutrition. Moreover, these two most important principles for facilitating a cure can by this method be extended over long periods of time without injury to the system.

20 East Sixty-Third Street.

DISCUSSIONS.

DR. FRIEDENWALD: Dr. Einhorn should be congratulated for having introduced this very effective form of treatment in ulcer of the stomach. I have utilized this method of treatment for many years in certain instances of ulcer, that is in severe forms associated with intense vomiting, nausea, recurrent hemorrhages, and especially those cases which have not been relieved by the usual methods of treatment. It has given me unusually good results in many instances, and I am confident that it has been a life saving measure in a number of cases. Of special interest are those cases to which Dr. Einhorn has called attention a few years ago; ulcers recurring after operation. These

cases are not infrequent, and I believe that before a second operation is contemplated, unless, of course, there be an obstruction or some other surgical complication, that this method of treatment should be undertaken, and that in many instances cure will be effected.

DR. BASCH: We know that the proper cure for ulcer has not been agreed upon yet, and we also know that with almost any method of therapy, or in spite of almost any method of therapy, ulcers have healed. As Dr. Bettman told us in summing up the argument this morning, it has been shown through autopsy that 50 per cent. of ulcers have healed without any treatment at all. I have used this duodenal tube method and I like it very much, and I used it in almost all types of ulcer, particularly duodenal ulcer, where there are no definite indications for surgical treatment. I recognize that there are hard and fast indications for surgical treatment, and I try to have surgical treatment where I can. Occasionally it happens that despite my advice, the patient insists upon medical treatment, and I am now and again surprised that cases with residue, cases with hard, indurated ulcerated conditions, heal after such medical treatment. One case particularly of 30 years' standing, another one of 20, that I recall just at this moment, who absolutely refused operation, have become clinically, and I believe even absolutely well with the institution of this method of treatment. The case of 30 years' standing was a matter of three years ago. I saw her last week again and she is perfectly well. This method, I think, approaches nearest to the surgical idea of ulcer treatment—I am talking about duodenal ulcer, and ulcers which cannot be removed by operation—than do any of the medical methods I know of. It gives absolute rest to the ulcerated area, and that is the first principle of surgical treatment of wounds, whether recent or chronic; perfect rest to the part. I am not, however, satisfied with two weeks' introduction of the tube. I feel that the length of time should depend upon the duration of the ulcer and the accompanying pathological factors. I think two weeks should be the minimum to expect an indurated area to begin to heal. I have had the tube in almost four weeks. I follow the cases up with a very definite and strict dietetic after-treatment. However, we must not be too optimistic in our results. Occasionally we will see a patient feel perfectly well six weeks or more and then suddenly we get a perforation or hemorrhage. But we must not, therefore, be discouraged, for in this method we have an ideal, almost surgical means, for the treatment of duodenal ulcer.

DR. EINHORN (closing): I could not speak of all the points; you see the time was limited to 15 minutes. Post-operative cases are greatly benefited by this method, and I also mentioned that I had a case of perforation of the duodenum after a gall-bladder operation. She got well after duodenal feeding. The tube went in a little further beyond the duodenum into the jejunum, and got perfectly well after two or three weeks of that feeding. Very extensive hemorrhages can be aided by the duodenal tube right at the time of the hemorrhage.

ADVANTAGES AND LIMITATIONS OF THE ROENTGEN RAY IN THE DIAGNOSIS OF GASTRO-INTESTINAL AFFECTIONS.

BY F. H. BAETJER, M. D., AND JULIUS FRIEDENWALD, M. D.,
BALTIMORE, MD.

The great difficulties arising in the diagnosis of many gastro-intestinal disturbances have long been recognized. In spite of our most thorough means of investigation including the use of the latest and most accurate methods which are at hand the diagnosis of many of these problems often remains obscure, and is indeed at times impossible.

With the advent of the Roentgen ray new light has been shed upon many of these intricate problems, and in consequence new and important facts in diagnosis are more frequently cleared up. While the Roentgen ray has been of the greatest aid in solving many of these important questions, and from what has been accomplished it seems quite probable that as the technic of roentgenology and our methods of interpretation of the X-ray plates become more perfected, that many unsolved difficulties in gastro-enterologic diagnosis will be cleared up; yet, it must be remembered that this method is only a single means of examination, and must not be relied upon alone, and that only when taken in connection with the other clinical signs does it attain its greatest value.

While the X-ray is of the greatest diagnostic help in solving difficult problems in gastro-enterology, there are limitations as to the conclusions which may be drawn from this method of diagnosis, and indeed in a small percentage of cases the results may at times even become misleading. This is not due, however, to the fact that the X-ray does not present actual conditions, but is caused by certain limitations in our technic as well as by the difficulty frequently encountered in the proper interpretation of the shadows on the plates.

Much has been written concerning the great value of the X-ray in the diagnosis of gastro-intestinal disturbances, but little has been brought forth regarding its limitations. It therefore seems timely that the limitations of this method of examination should

be definitely noted, for while we fully recognize that many of these difficulties will in time be overcome, yet at present we are anxious to emphasize the fact that in this method we possess but a single form of examination which cannot be relied upon alone, but when taken in connection with the other clinical signs is of the greatest diagnostic help.

ULCER OF THE STOMACH AND DUODENUM.

It is a well-known fact that the Roentgen ray is of the highest diagnostic aid in revealing ulcers of the stomach and duodenum, and yet in some instances these lesions are not revealed by this method of examination or defects may be present, which are interpreted as ulcerations which cannot be detected at operation. In explanation of these facts it might be said that ulcers situated in certain locations in the stomach and duodenum may not be revealed by the X-ray. Only when the lesions are situated on the anterior surface of the stomach and along the anterior surface of the lesser and greater curvatures are they revealed. Yet, according to our observations the functional signs are often as important as the presence of a defect at arriving at definite conclusions as to the presence of an ulcer. It is, therefore, of the greatest importance to bear in mind the value of the functional signs in diagnosis, even though a defect or deformity may be absent in repeated examinations. Thus in eight per cent. of cases studied by us, there were no defects observed, yet the functional activity of the stomach pointed definitely to ulcer. However, in a certain number of cases there are definite clinical evidences of ulceration, and yet the X-ray presents no indication whatsoever of this condition. This may be due to adhesions, to which attention will be called later on, masking the true condition, but the absence of definite X-ray signs cannot always be traced to this factor.

It happens at times that a complicating appendicitis or cholecystitis may be so marked as to mask any findings in the stomach itself, and the significance of a small filling defect might easily be overlooked.

In one of our cases (Dr. B.) a man of sixty-four years of age, who had presented definite clinical signs of ulceration for years, and in whom the X-ray findings had been positive four years previously, was again examined. The X-ray examination showed

a mass of adhesions in the upper right quadrant region, involving the duodenum and gall-bladder. The duodenum was fixed and deformed. The X-ray diagnosis was that of adhesions, and it was impossible to determine whether the deformity was due to an ulcer or to adhesions. The operation showed a large duodenal ulcer with adhesions.

In another instance (Mrs. B.), the X-ray findings pointed to a chronic appendix with a very spastic, irritable stomach, and no defects could be made out. Consequently, the X-ray conclusion was that the condition of the stomach was simply reflex, but at operation, besides a chronic appendix, there was found an ulcerative lesion in the duodenum.

In a large series that were examined, in three per cent. of the cases of ulceration no filling defects could be made out by means of the X-ray examination; and in 15 per cent. of the cases the symptoms were not definite enough to make an absolute diagnosis of ulcer.

It is especially in those instances in which there are marked evidences of chronic appendicitis or cholelithiasis with adhesions that ulcer of the stomach or duodenum may be present, and yet not be revealed. On the other hand, one cannot at this time pass by another group of cases in which the ulcerations are of the so-called mucous type. These are easily demonstrated by means of the X-ray, but are rarely observed at operation unless the stomach or duodenum are actually opened. It has been our experience in a number of instances of this kind that when an exploratory operation was performed, and the stomach and duodenum palpated, no ulcer was observed; the symptoms nevertheless would persist and these cases would later present such definite signs that for the relief of the symptoms a second incision was indicated when the ulcer was revealed. In one such case (Miss F.), the operation revealed absolutely nothing abnormal. The patient was temporarily relieved, but returned at the end of six months, presenting all of her former symptoms. A second X-ray examination disclosed almost a complete pyloric obstruction. The second operation exhibited a large indurated ulcer in the pylorus with scar formation. The former examination had pointed to ulcer at the pylorus, and since it could not be palpated at operation it was overlooked. There cannot be any question but that many ulcerations of this type are passed by unnoted at operation.

The greatest difficulties arise in the diagnosis of complicated cases; that is, when adhesions are present, due either to the healing of the ulcer or to inflammations connected with one or other of the organs in the abdominal cavity. These adhesions so frequently mask the usual findings, that it is often impossible to determine whether we are really dealing with an ulcer or whether a lesion of some other organ is causing the symptoms. Such adhesions may arise from the ulcer itself, from the gall-bladder or appendix, or there may be no adhesions at all in the region of the stomach, and the gastric findings may be due to a purely reflex condition or to spasm.

It is necessary at times, in a certain proportion of cases in which adhesions of the stomach are present, to lay special stress upon the clinical history of the case before a final decision can be made. If the clinical signs coincide with a definite history of ulcer, we must conclude that the adhesions have taken their origin from the ulcer; if of cholelithiasis, from the gall-bladder; and so on, in as much as from a roentgenological point of view the appearances are frequently identical. In nine per cent. of the cases in our series the diagnosis was rendered doubtful by the presence of adhesions.

As we have frequently pointed out, unless due care be exercised, one may be misled by certain reflex or spastic conditions of the stomach. It is not uncommon to observe a stomach presenting a persistent filling defect in a definite area and continuing over a period of an hour or two. In one instance under our observation it persisted over a period of 48 hours. In this particular case the filling defect was so large and so definite, notwithstanding the fact that the clinical evidence was negative, that the case was used for repeated demonstrations of the typical ulcer. Operation, however, revealed a perfectly normal stomach. In such cases the spasm of the stomach can easily be eliminated by the administration of full doses of atropin for one or two days until the patient is well under its influence. A second examination made under these conditions will generally show that the suspicious area has completely vanished, and that the defect was in reality a spasm.

When ulcerations are situated at or near the pylorus, important aid is rendered in diagnosis by the presence of obstructive signs produced by the cicatrix while the ulcer is healing. These signs

are not only revealed by the X-ray examination but also by clinical methods. It occasionally happens, however, that even though the clinical history as well as the retention meals point definitely to obstruction, that this condition may be overlooked by the Roentgen ray examination.

In a patient, Mrs. C., in whom the clinical signs and retention tests pointed definitely to obstruction, the X-ray gave no evidence of this condition. Levy, too, has recently pointed out that the Roentgen ray as it is commonly employed is at times not as delicate in determining gastric motility as the usual retention meals. On the other hand, there are many instances of partial obstructions in the early stages of gastrectasia where the obstruction is still incomplete in which the diagnosis is often very difficult. The clinical signs of this condition appear so intermittently, that they are overlooked, while on the other hand this condition can be definitely determined by X-ray examination.

CARCINOMA OF THE STOMACH.

The Roentgen ray diagnosis of carcinoma of the stomach is not always positive, especially in the early stages, as the lesion may be mistaken for simple ulceration.

In cancer of the cardiac area the stricture and filling defect may be absent in early lesions, and the condition may be overlooked on account of the great difficulty in properly filling the cardiac end of the stomach.

In lesions in the body of the stomach, one may have an extensive growth, and yet the patient may complain of very few symptoms, in as much as the motor activity is not interfered with.

As this lesion does not affect the cardiac or the pyloric orifice, obstructive symptoms are absent, the stomach emptying at the normal rate or perhaps a little more rapidly than normally. The Roentgen ray examination of the stomach of this form generally shows that the peristaltic waves are interrupted in their course at the seat of the lesion, inasmuch as the lesion itself is hard and indurated, and does not admit of further peristaltic movements. In addition, a persistent filling defect is almost always present. In the third class of cases, that is, in lesions about the pylorus, the symptoms arise early as a rule, and are due to a partial obstruction to which attention has already been called.

From a roentgenological point of view we classify growths at the pylorus into two forms.

The first, the annular carcinoma, has its origin within the pylorus itself, and although it may be very small in size, produces symptoms relatively early, as it soon causes a greater or less degree of obstruction.

The second type also has its origin at the pylorus; it is not annular in form, but invasive, most frequently extending along the wall of the stomach, especially at the pylorus. Such a growth may or may not produce early obstruction.

The annular type of carcinoma is easily recognized, inasmuch as the stomach is observed to be perfectly normal except at the pyloric end. The pylorus, instead of being clear-cut and sharp, is generally somewhat thickened, and slightly depressed in the center, so that the structure assumes the shape of a crater.

The second type of carcinoma may exist for some time without being recognized, because in the absence of any symptoms of obstruction the patient, being still in a comfortable state, does not present himself for an examination. In this type of growth one finds a persistent filling defect in or close to the pylorus, and ascending away from it. The mass is indurated and free from peristaltic waves.

Notwithstanding the very definite X-ray signs of gastric carcinoma, there are instances especially in the early stages when this condition is not recognized. This is due to the fact that the defect is so small as to be entirely overlooked, and the functional activity of the stomach is not interfered with.

On the other hand, unless due care be exercised, spastic or reflex conditions may lead to errors in diagnosis. It is not uncommon to observe a large persistent filling defect in a definite area simulating carcinoma and continuing over a period of hours. In one case this defect persisted constantly without intermission for ten days, and was only found to be a spasm at operation. In a second instance the roentgenological examination was made by one of our well-known roentgenologists with a diagnosis of carcinoma. Three months later the patient was seen by us, and the roentgenological examination showed identically the same condition as was present at first. The patient was given atropin in full doses, and when the second examination was made, the filling defect had completely disappeared. The patient, however,

not being content, insisted upon an exploratory operation, which revealed a perfectly normal stomach.

In the X-ray study of gastric carcinoma the differential diagnosis between this condition and ulcer is often very difficult. It is still a much debated question as to whether carcinoma of the stomach has its origin primarily as such, or is the result of a transition from an old ulcer. If the latter view be correct, one can readily understand the difficulty in determining when the benign condition begins its transition into malignancy. The situation of both carcinoma and ulcer is very frequently the same, although we observe carcinoma more frequently on the greater curvature than ulcer.

In the differential diagnosis between the two conditions, the points to be taken into consideration are as follows:

1. *Peristalsis*.—In carcinoma, unless there is obstruction, there is always hypermotility with rapid evacuation of contents. In ulcer there is always hypermotility, with a spasm of the pylorus and more or less retention of contents.

2. *Position*.—Carcinoma may occur in any part of the stomach. The invasive lesions are more frequently seen on the lesser curvature near the pylorus, and less frequently on the greater curvature. The massive growths are more generally seen on the greater curvature, whereas ulcer is generally observed on the lesser curvature near the pylorus, although it may occur on the greater curvature.

3. *Filling Defect*.—In carcinoma the filling defect is generally surrounded by an invasive area, which, while not appearing on the plate, interferes with motility, producing an apparently large dead area. In ulcer the filling defect is much smaller, and is not so apt to have the immediate peristaltic waves interfered with, although, if the inflammatory area be large, there may be also a dead area surrounding the filling defect.

Carcinoma of the pylorus in the earliest stage is generally annular and produces a crater-like appearance. In ulcer of the pylorus there is a filling defect, but it does not generally assume the crater-like appearance.

When any of these conditions pass on to the obstructive stage the change that is caused by the dilatation may mask the signs associated with the filling defect.

In our experience in the very early stages of gastric cancer it

is frequently impossible to determine whether we are dealing with a malignant or a simple ulceration. Our main aim, however, is to decide whether the lesion at hand is really an ulcer or not. Inasmuch as indurated gastric ulcers have at times a tendency to become malignant and produce roentgenograms similar to those which are cancerous, they must be included in the same class. The exact diagnosis must be cleared up by further investigation into the clinical history and the examination. But even under these conditions there are many instances in which the diagnosis may still remain in doubt until operation and microscopic examination of the specimen after removal finally establishes the true nature of the disease.

GALL-BLADDER DISEASE AND GALL STONES.

The Roentgen ray indications of gall-bladder infections are revealed by the presence of adhesions dragging the stomach and duodenum over in the direction of the gall-bladder. The adhesions, however, need not necessarily extend from the stomach and duodenum, but may arise directly from the gall bladder itself extending to various parts of the intestinal tract, and are apt to produce varying degrees of stasis, retention and obstruction according to their extent, and thus give us a definite picture in the plates as to the chronicity of the gall-bladder disturbance. Frequently this condition is associated with a filling defect in the duodenum, and a deformity of the duodenal cap. When that condition arises it is frequently impossible to determine whether we are dealing with a primary gall-bladder or duodenal disease, and it is only by a study of the clinical history, as we have already pointed out, that a correct diagnosis can be made.

The demonstration of gall stones by the Roentgen rays has itself reached a position that warrants its more general use as an aid in diagnosis, and if the gall-bladder region is examined for stones prior to the bismuth examination of the gastro-intestinal tract, gall stones may be absolutely demonstrated in a certain percentage of the cases, depending largely upon the amount of calcium salts present. Gall stones composed entirely of pure cholesterin are practically invisible to the ray. It is difficult to give percentages, as they vary so markedly, according to the type of case and the operator, that figures would be misleading. One must remember, however, that a negative diagnosis does

not rule out gall stones. The calcium mixture is the only part shown by the X-ray, and the clearness of the gall stones upon the plates will be proportionate to the amount of calcium. In as much as in a large proportion of patients affected with gall stones for some time the stones contain calcium, it is more than likely that as the technic becomes more perfected, gall stones may be demonstrated in many instances, especially where the symptoms have been of long duration.

There are sources here, too, however, of error, the main ones being renal calculi, calcified mesenteric glands and costochondral ossifications. There have been a number of instances in our experience in which shadows were diagnosed as gall stones, which were not found present at operation.

CHRONIC APPENDICITIS.

The Roentgen ray examination of the appendix itself in its chronic state is capable of rendering valuable service in a certain number of instances. When the lumen of the appendix is closed as the result of an alterative process, bismuth will fail to enter.

We have frequently pointed out how partial obstructions of the pylorus are apt to lead to the production of adhesions extending to the lower right quadrant dragging the stomach in that direction, and these are often associated with chronic disease of the appendix. Adhesions associated with the appendix, however, need not extend from the stomach or even from the duodenum, but may arise from the appendix itself or its surrounding structures and may lead to a varying degree of cecal or ileal stasis or even to partial colonic obstruction.

With proper technic the appendix may be visualized in many instances, whereupon roentgenographs of this organ may be made.

Case recommends a special technic for the examination of the appendix. "1. The necessity of examining the patient in the horizontal position with the screen held over the abdomen, and the tube underneath the table. 2. The necessity of palpating the abdomen under the screen, with the gloved hand or preferably with the wooden spoon, and noting the localized tenderness. The time of the examination is of some importance. Shortly after the cecum begins to fill, the appendix also fills. This usually occurs in six hours, although there are many cases in which the cecum is filled earlier or later. From this time on until the bowel

is empty, and frequently for some time afterward, the appendix remains visible.

"The ordinary technic of roentgenology with the patient standing or lying, is not likely to show the appendix; we must first find the appendix fluoroscopically, and then make the roentgenograph. When the appendix remains visible for more than a day or two after the bismuth examination, it is in proportion to its poor drainage, a dangerous appendix."

There are certain instances in which the bismuth shadows persist for many hours or days, and sometimes weeks after the bowel has been emptied of its bismuth.

While the X-ray renders valuable service in diagnosis in many instances of chronic appendicitis, there are a certain number in which it does not demonstrate this condition, or where its results may even be misleading. This may be due to the fact that on account of its unusual position the appendix cannot be visualized or, as has already been pointed out, that its lumen is closed due to an obstructive process so that the bismuth is prevented from entering or finally to the presence of mild adhesions which may not be revealed in the plates.

Attention must also be called to a potent source of error which should always be borne in mind. As a reflex condition a chronic appendicitis may give rise to a picture somewhat similar to that observed in duodenal ulcer; that is, there is gastric and duodenal hypermotility with a definite filling defect, and deformity of the duodenal cap; if this picture is rather marked, and the appendiceal adhesions are slight one can readily understand how chronic appendicitis might be mistaken for a duodenal ulcer, and in fact this error is sometimes made. In instances of this kind the plates must be most carefully studied, repeated examinations made if necessary, and in most instances a combined fluoroscopic examination, study of the plates, together with the clinical history will point to the correct diagnosis; but even at times after a most careful investigation in every direction, the correct diagnosis may still remain in doubt.

INTESTINAL ADHESIONS AND ANGULATIONS.

When the bismuth meal is taken it ordinarily reaches the cecum in from seven to ten hours, the transverse colon in twelve, and the sigmoid and rectum in eighteen to twenty hours. Delay in

the passage of the bismuth may be due to the various conditions; to a dilatation and retention in the cecum, to ptosis of any portion of the colon, especially the transverse colon with intestinal stasis; to adhesions in various portions of the intestinal tract, including the gall-bladder region, cecal region, splenic flexure or sigmoid or to a redundant transverse colon or sigmoid, to ileal stasis or frequently to angulations at the hepatic or splenic flexures, and finally to carcinoma of the colon. Carcinoma of the colon may exist for a long period of time before obstructive symptoms are noted. Filling defects are frequently observed in this condition, but great care must be exercised to be positive of the constancy of these findings, both by fluoroscopic examination and by means of bismuth enemata.

In a special instance which has come to our attention in which a capable roentgenologist made a diagnosis of carcinoma of the ascending colon, and in which the diagnosis was confirmed by all the methods usually practiced by roentgenologists, the filling defect and partial obstruction was found due to the dragging down of the bowel by adhesions connected with an inguinal hernia. In as much as adhesions are detected only by changes, and delays in motility, great care must be exercised in drawing conclusions regarding these findings.

Spasmodic contractions can usually be eliminated by the free administration of atropine or belladonna and in doubtful cases repeated examinations must be made. There can be no question but that frequent errors are made in the diagnosis of intestinal adhesions, which are usually due, however, to a lack of proper technic or insufficient observation.

We have attempted in a rather imperfect way to direct attention to the fact that as yet the results of roentgenological diagnosis are not sufficiently perfected in all cases to be relied upon alone. While the X-ray is probably our one greatest aid in diagnosis, yet it is only one method of examination, and the best results are obtained by the combination of it, and a thorough clinical study.

DISCUSSIONS.

DR. LICHTY: I believe it was about ten years ago when Dr. Freidenwald and Dr. Baetjer read a paper on the value of X-ray examination in gastro-intestinal work, and I believe if we compared that paper with the paper which was read today, we would possibly strike the trend

which X-ray work has taken in gastro-enterology. In other words, Dr. Friedenwald is not as optimistic on X-ray examinations today as he was several years ago. I believe that coincides with my experience at least, and especially so in the past year. As Dr. White has brought out, work of this kind will have to be done very carefully. It must be done again and again, and time and expense for that matter must not be an element to enter into the examination. There must be a free and thorough examination; unless it is done we are likely to be misled, I remember several years ago when I was called in to decide as to whether an operation was to be done or not, the X-ray showed a diverticulum in the lesser curvature of the stomach which was supposed to be a perforating ulcer. I did not insist upon a repetition of the X-ray and rather leaned toward an operation. An operation was done and the stomach was found to be normal.

Carcinoma of the bowel: The carcinoma I have in mind is one that was found in the sigmoid. The patient gave us a very definite history of bloody discharge and she thought that she had passed a small mass from her bowel. The gastric analysis was normal; the stool was normal and yet the history was definite. We had an X-ray examination made. It was negative. We had a bismuth enema, and it was negative. I then asked a gynecologist to make a vaginal examination, and he was able to palpate a small mass on the left side above the ovary. He operated on the strength of this and found a small carcinoma of the sigmoid. Now, when such things can happen we must be very careful with X-ray interpretation. Another point to which I would like to call attention, a point that Dr. Friedenwald has presented today, is the factor of adhesions of the omentum to any part of the abdominal cavity. I believe that this can simulate any pathology in the gastro-intestinal canal. I have seen the right side of the omentum attached to the caecum and a very definite diagnosis of carcinoma of the pylorus made, not only on the X-ray alone, but on other findings. When the abdomen was opened—it had been an old appendix case—the omentum was adherent as described. So I think it takes time and patience if we are going to utilize the X-ray examination in gastro-intestinal work. I believe that Dr. Friedenwald and Dr. Baetjer have given us a paper which we should take into consideration.

DR. MILLS: I really can't add to the discussion of Dr. Friedenwald's paper. I am entirely in accord with everything that he has said, and everything those who discussed the paper have said. It has been my hope to help to bring the subject of gastro-intestinal roentgenology to your attention. A year or two ago I was just a little depressed; it seemed to me that some were not catching the ball; but I have certainly no reason to think that the ball has not been caught now. Certainly the attitude towards gastro-intestinal roentgenology has changed. It may have been that the X-ray men overdid the thing and made contentions that were not justified; it is even possible that clinicians were not as broadly receptive as they might have been.

There is a new shadow ahead. General roentgenology has in a way served its greatest purpose, that of introducing the subject. While we will probably never see the time that we have not need of general roentgenologists, I do not believe that the highest expression of roentgenology finds its outlet through general roentgenology but in specialized work closely associated with clinical medicine, a fact that is being appreciated by the leaders among the X-ray men themselves.

DR. BASCH: Dr. Friedenwald has taken a very conservative and proper stand upon this question, and I have nothing to add to what he has said, excepting that he has not pointed out the advantages of roentgenology in the rather unusual conditions which we meet with, such as diverticula, cysts, particularly pancreatic cysts, pancreatic stones, diaphragmatic hernia and benign tumors of the stomach. These conditions, though comparatively rare, are very important, because the cases very often puzzle us, and if we imagine roentgenology only good for ulcers and carcinoma, we will often be surprised to find an unusual diagnosis made by the X-ray. I agree thoroughly with Dr. Friedenwald that in early diagnosis of carcinoma roentgenology is not as good as the mass of roentgenologists would have us believe. Once in a while we find there it is not of much value in the advanced cases of carcinoma. I am not speaking of a clinician's work, but that done by an A-1 roentgenologist. I have in mind two cases of carcinoma of the stomach that were inoperable by reason of metastasis in the liver, which were reported as negative by the roentgenologist. I would also corroborate the negative result in tumors of the lower intestines. There, too, I have had cases reported negatively that upon operation have shown neoplasms. In one case, just a few months ago, one could feel the neoplasm. The roentgenologist reported it negatively. Proctoscopic examination showed an ulcerated area, and the physician in charge diagnosed the tumor through ordinary clinical methods. One great advantage of roentgenology is that it would help us classify our gastric and other cases into the surgical or medical.

DR. FRIEDENWALD (closing): I wish to thank the gentlemen of this society for their very thorough discussion of this paper. I only regret that Dr. Baetjer is not here to help us discuss it.

HYSTERICAL VOMITING.

BY ARTHUR F. HURST, M. A., M. D., OXON., F. R. C. P.,

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LONDON.

During the past three years my time has been almost exclusively occupied in the care of soldiers suffering from war neuroses. I have been led by my experience to propose a new definition of hysteria. All previous definitions have presupposed the existence of certain mental and physical stigmata, which were thought to be present before the symptoms developed and to persist and require treatment after the disappearance of the symptoms. I believe, however, with Babinski, that physical stigmata never exist until they have been produced by the unconscious suggestion of the observer; and the mental stigmata, such as an abnormal degree of suggestibility, are only present in a comparatively small proportion of cases. Consequently, if we only look for hysterical symptoms in individuals who, according to the old criterion, would be regarded as hysterical, we shall miss numerous cases in which there is nothing in the individual's mental characteristics which would indicate that he would be likely to be suffering from hysteria.

My definition is as follows: Hysteria is a condition in which symptoms are present, which have been produced by suggestion, and are curable by psychotherapy.

Having accepted this view of hysteria we began to look for cases of hysteria in the medical and surgical wards of hospitals among patients whose disabilities had been diagnosed as organic in nature. Among other discoveries was that of the remarkable frequency of hysterical vomiting. As every type which we met had its counterpart in civil life, a description of the origin of these cases and of the unexpectedly successful treatment which we gradually developed may be of interest even now that the war is over. A preliminary note on the subject was published by Captain W. R. Reynell, in the *Seale Hayne Neurological Studies* for November, 1918.

ETIOLOGY.

In every case the hysterical vomiting was suggested by the existence of some form of vomiting which was not in itself hysterical.

Every variety of non-hysterical vomiting may be perpetuated and exaggerated by suggestion after its original cause has disappeared.

The causes of vomiting may be classified as follows: 1, local; 2, reflex; 3, toxic; and 4, central.

The first group includes the vomiting which arises from irritation of the stomach. The irritant produces a varying degree of inflammatory reaction, and the gastritis which results is associated with vomiting, which has for its object the removal from the stomach of the irritant which gave rise to the gastritis. Such cases are very common in civil life, the most important form being that due to food poisoning. There were a very large number among our cases that resulted from gassing, the irritant gas having called forth an abundant secretion of saliva in which some of it was dissolved and swallowed. That actual gastritis was produced was proved in a number of post mortem examinations, and by the presence of abundant mucus together with traces of blood in the vomited material during the first two or three days. In the vast majority of cases, both in civil life and in war, vomiting ceased after two or three days, or at latest by the end of a week. In rare instances the acute gastritis is followed by chronic gastritis, but vomiting is not a characteristic symptom of this. It may, therefore, be confidently presumed that if vomiting persists for more than a fortnight after the onset, it is no longer a direct result of the gastric irritation, but is due to the perpetuation of the symptom by suggestion. Consequently, it is now hysterical, and instead of requiring treatment with restricted diet, lavage and drugs, it should be rapidly cured by psychotherapy.

All of the numerous cases of hysterical vomiting following gassing which came under our care had already been in other hospitals for many months, and the vomiting had been diagnosed as due to gastritis, although no other symptom was present, and the vomited material contained no mucus or other abnormal constituent. A few had been diagnosed as gastric or duodenal ulcer, and one patient had even been subjected to a gastro-enterostomy. Several men had been invalided from the service, but no improvement had followed their return to civil life. Without exception they all recovered rapidly under psychotherapy and were able at once to take ordinary food and lead a normal life in spite of having been on a strict and often purely fluid diet for many months, and, in some cases, having been kept in bed during the entire period.

The constant vomiting which is such a common symptom in anemic young women, and which was formerly regarded as evidence of a gastric ulcer, is in the vast majority of cases hysterical. It is often associated with pain which may make the simulation of gastric ulcer very close, but the vomiting is more frequent than is generally the case in a gastric ulcer, unless the latter has led to pyloric obstruction. The onset generally dates from some acute attack of gastric irritation caused by food poisoning; but the vomiting, instead of ceasing within twenty-four hours, as it would under normal conditions, continues indefinitely as a result of autosuggestion. Dieting has no effect upon it, as many of these patients continue to be sick for many months in spite of a diet of slops, and I have even seen it continue after rectal feeding had been resorted to. A striking feature in these cases is the absence of wasting, as in the majority of instances the patient remains unexpectedly plump. Directly she is taken away from her own home surroundings into a hospital and given a full diet the vomiting ceases. A certain amount of moral persuasion is often required, but if the patient continues to vomit she must be given the same meal over and over again until she ceases to do so. It is very rare in such cases for a symptom to persist for more than twenty-four hours. At the same time the epigastric tenderness, which is often very well developed, probably as a result of the suggestive effect of frequent examinations, should be entirely neglected, as, if not looked for, it will spontaneously disappear.

It seems probable that many cases of persistent vomiting after operations are purely hysterical, and the well-known efficacy of gastric lavage in such cases may be more the result of suggestion than due to the removal of mucus or other irritant from the stomach. On several occasions patients who have suffered so severely from vomiting after previous anesthetics that they have only been persuaded with difficulty to undergo a further operation, have not vomited at all when told convincingly that the anesthetist, who was going to give them ether on this occasion, had a special method which was certain to prevent vomiting, although it was really quite indifferent what special modification he used.

In the following case the vomiting caused by an attack of dysentery in infancy was perpetuated as hysterical vomiting asso-

ciated with hysterical anorexia, and persisted until it was treated by rational psychotherapy six years later.

CASE.—John M., born in India in 1913. He was weaned when four months old, but great difficulty was experienced in persuading him to take the bottle, even with the assistance of a trained nurse. When a year old he nearly died of dysentery. After his recovery from the acute symptoms he continued to vomit all solid food. This condition persisted after he was brought to England in 1915. His mother, a highly strung neurotic woman, spent a considerable part of each day in feeding him. He disliked all food, and could be persuaded to take anything into his mouth only with extreme difficulty. Unless it was fluid he kept it between his teeth and cheeks for an indefinite period. He invariably vomited solid food, and was only able to keep down rusks and milk pudding after they had been pushed several times through a sieve. Each meal was the scene of unpleasant struggles, in which he often kicked and screamed.

His mother had no control over him and gave way to every whim. When I first saw him in May, 1919, the condition had not improved. He still vomited part of every meal, and spent the comparatively short intervals between his very prolonged meals churning between his teeth and cheeks any semi-solid food that he had taken. In spite of this he was very well nourished, though flabby through want of exercise. He could not even be persuaded to take sweets by himself and invariably had to be fed; although in other ways he was beyond the average in intelligence he was extremely awkward with his spoon, knife and fork.

As the condition was clearly one of hysterical anorexia and vomiting, I decided to attempt to treat him in the same way as one would treat an adult with similar symptoms—by explanation and persuasion. At the first attempt, after much protest and some weeping he suddenly settled down and ate an ordinary meal of meat and vegetables, followed by pudding, in a reasonable time, without subsequent vomiting. I had no opportunity of seeing him again, but arranged for him to be separated from his mother and looked after by a nurse. He was now quicker than before with his meals and did not vomit again, but the great success of the meal he ate in my presence was not repeated, as he took quite an hour for each of his meals, after each of which he still kept some food in his cheeks, particularly bread and pota-

toes. As no further progress occurred, Dr. C. H. Ripman looked after him for some weeks; he was given ordinary meals and was firmly encouraged to eat them. If he took longer than was considered reasonable (twenty minutes for breakfast and half an hour for lunch), or if he cried, he was threatened with a further portion, and, if necessary, the threat was carried out. After three weeks he was eating normally, but, though not otherwise clumsy, he was still awkward in the use of spoon and fork.

He had never been taught to help himself, and was very dependent on other people in every way, but on being separated from his mother he soon became more self-reliant, and dressed and undressed himself. His mother had always sat in his bedroom until he was asleep, saying he could not sleep otherwise, but he quickly learned to sleep in her absence.

After returning to his mother he remained quite well for a few weeks, but then began to eat rather more slowly, and one day he vomited. His mother was alarmed, and took him at once to Doctor Ripman, with the result that he made only one further half-hearted attempt to vomit and in a short time he was taking his food quite normally again. He has now (October, 1919) been sent to school, and no further difficulty has been experienced in getting him to eat.

By reflex vomiting is meant vomiting which originates by a reflex from some organ other than the stomach. The most common varieties are due to abdominal diseases, such as appendicitis, and phthisis. We saw examples of these conditions in which the frequency of the vomiting had been greatly exaggerated as a result of autosuggestion, and some in which the vomiting had persisted after complete recovery from the primary cause had occurred. There is obviously no difference between a case of this type in soldiers and in civilians, and I am convinced that the frequency of the association of hysterical vomiting with reflex vomiting has not in the past received the attention it deserves. We found that treatment of the vomiting by psychotherapy in cases of phthisis resulted in such a diminution in frequency of the vomiting that the patients, instead of vomiting after every meal, might only vomit once or twice a week. Consequently the weight rapidly increased and the general health greatly improved. In a number of cases in which general abdominal discomfort was present and the exact diagnosis was at first uncertain, the

associated vomiting was almost completely removed by psychotherapy. It was then found that the very infrequent vomiting which still persisted was due to the presence of chronic appendicitis. On the removal of the appendix the vomiting finally ceased.

On the other hand, we saw several cases in which appendicectomy had been done, and the patient still vomited. The persistent vomiting was hysterical and removable by psychotherapy, and it is probable that if similar treatment had been given before the operation the latter would have proved completely successful. If the possibility of the association of hysterical vomiting with other symptoms of chronic appendicitis is recognized and suitable treatment is given, disappointing results of operations would be much less frequent. In the same way, the vomiting due to other organic conditions, such as gall stones, gastric and duodenal ulcer, may be in part hysterical, and when vomiting persists after the removal of gall stones or after the medical or surgical relief of an ulcer, this possibility should always be borne in mind. I believe, for example, that some of the rare fatal cases which are caused by persistent vomiting after gastro-enterostomy are hysterical, and death could have been averted by psychotherapy.

Seasickness is another form of reflex vomiting, the reflex arising in the semi-circular canals. Normal individuals vary greatly in the irritability of their semi-circular canals. Thus an otherwise healthy man vomited for several hours after testing his vestibular reaction by turning round four times with his back bent so that his forehead rested on a walking stick placed vertically on the ground. Consequently some people vomit much more readily than others when at sea. In most cases the tendency to seasickness is absent in infants, and in some it diminishes during a long voyage and also with advancing age.

It is, I believe, extremely common for bad travelers to develop into still worse ones as a result of auto-suggestion. So convinced are they that the shortest sea journey will make them ill that they make preparations for being sick directly they get on board. It would be difficult for anybody to resist the suggestion of a crowd of people lying down in a stuffy cabin of a channel steamboat, each with his basin ready, and many beginning to vomit even before the boat left her moorings. The most extreme case I have seen was an old lady who began to vomit in the train to Dover, en route for the continent, although she never suffered from train sickness apart from this.

Psychotherapy by encouragement and explanation will often prevent a passenger from being ill, although he may be convinced from past experience that he could never stand a sea journey. Gross suggestion may act with equal success. This is the only possible explanation of the remarkable results achieved by an American naval medical officer recently recorded in the *Journal of the American Medical Association* as well as in the lay press. He almost completely prevented seasickness on several transports full of troops crossing the Atlantic by stuffing the men's ears with cotton wool in the hope of damping the vestibular reflex. But it is quite obvious that no treatment of the external ear could influence the effect of movements of the head on the semi-circular canals, so that the results obtained must have been due to auto-suggestion on himself and heterosuggestion on his patients.

The most common example of toxic vomiting is the vomiting in certain acute infections, such as scarlet fever and influenza. If vomiting persists after recovery from the infection, it is in all probability hysterical. We saw some cases of vomiting following trench fever, dysentery, paratyphoid fever, and influenza in soldiers. The so-called pernicious vomiting of pregnancy has generally been regarded as toxic in origin, but in my opinion it is invariably hysterical. The vomiting which is physiological during the first few weeks of gestation, may be reflex or toxic in its origin; but if it persists after this its perpetuation is due to auto-suggestion, and it can invariably be cured by psycho-therapy. The chemical changes in the urine pointing to acid intoxication and the other supposed evidences of toxemia are entirely due to the starvation which results from the severity of the vomiting, as these symptoms disappear directly the vomiting is cured by psychotherapy. In 1915 I saw a lady, twenty-one years old, who had been vomiting persistently during the first five months of pregnancy, and who for many weeks had been unable to keep even fluid food down; during the last three weeks she had been fed by rectum, but continued to vomit fluid. Her urine contained diacetic acid, acetone, and a trace of albumin, and her breath smelled of acetone. A gynecologist, who saw her with me, recommended immediate emptying of the uterus. I asked for twenty-four hours' delay, and had a talk with the patient at once. I did not leave her until I saw she was convinced that what I had told her was true—that her vomiting was nothing more than the

perpetuation by an idea of what was natural at first, and that as the cause of her vomiting was no longer operable, she would be able to eat her ordinary dinner that evening and would not vomit again. After my departure she ate the first solid meal she had taken for six months, and did not vomit again; her child was born at full term without any recurrence of the vomiting.

Central vomiting may be due to organic nervous disease, such as cerebral tumor or meningitis. This is obviously of no importance in connection with hysteria. But there is another variety of central vomiting which arises as the physical expression of an emotion and which is of great importance. Extreme disgust and occasionally an emotion such as fear may give rise to vomiting. This vomiting, though emotional and therefore nervous in origin, is not in any way hysterical, as it is not produced by suggestion, being the natural result of the emotion. If, however, it persists after the emotion is no longer operative, its perpetuation is due to auto-suggestion, and it is then hysterical and therefore curable by psychotherapy. We saw a number of cases in which the vomiting began after some particularly disgusting experience at the front. In one case it developed as the result of fright caused by being torpedoed. Similar cases occur in civil life, and such a possibility should always be considered in cases of vomiting, in which no other abdominal symptom is present and in which the origin is completely obscure; a thorough cross-examination into the history will often show that the vomiting began immediately after some emotional crisis.

The following is a very good example of severe vomiting following vomiting of an emotional origin. In this case, as in many others of the kind, the origin may not at first be clear, but becomes apparent when the patient's confidence has been obtained and a complete history is elicited. The wife of an officer who had gone out with the original Expeditionary Force in August, 1914, had naturally been living in a condition of severe strain. In the summer of 1915 for the first time she saw him off from Southampton instead of from London when he was returning to France from leave. The carriage was crowded and hot, and immediately after the train journey was over she had a severe attack of vomiting. From that moment she vomited whenever she traveled by train although she had never done so before. She therefore traveled whenever possible in a car, but soon this also caused

vomiting. After a time she found she could not go into a public place, such as a theatre or a church, without vomiting, and finally she was unable to leave her home at all. On each occasion when her husband returned on leave she was unable to go anywhere with him, as every time she attempted anything the vomiting returned. She had all sorts of treatment for her stomach but without avail. I first saw her in August, 1918. It soon became clear that she had attempted to explain away the vomiting, which on the first occasion had been clearly of pure emotional origin by trying to make herself think it was due to the journey in a hot and crowded train. As a result, any vehicle or overcrowded building had later on the same effect on her. As soon as this was explained to her and she was made to realize the purely psychical origin of her vomiting, it ceased, and the numerous other nervous symptoms which had developed simultaneously gradually disappeared.

DIAGNOSIS.

The possibility of hysterical vomiting should always be borne in mind when it arises under such circumstances as described above. It should be particularly suspected when no other abdominal symptoms are present as, although a certain amount of abdominal discomfort may be associated with the vomiting, it is often absent, and actual pain is uncommon. A careful abdominal examination should be made, but when some definite organic disease is found, if the vomiting is very persistent and severe the possibility that it is in part hysterical should be remembered, as otherwise the symptoms may persist, although to a diminished degree, after the organic disease has been removed by medical or surgical treatment. A more complete investigation by means of gastric analysis and X-ray examinations should always be made in doubtful cases, but when from the history it is obvious that the condition is hysterical nothing can be gained by such an examination, as in my experience the patient is always satisfied with a plain abdominal examination. A more thorough examination instead of helping to relieve his mind, may have the contrary effect of making him think that the physician believes there is something serious the matter with him, and when told that this is not the case he may believe dreadful things are being hidden from him. Moreover, the discovery of some slight increase or

diminution in gastric acidity may lead to the prescribing of diet and drugs to correct this, or the skiagram may show some slight delay in evacuation, atonic dilatation or ptosis, and lead to the recommendation of further dietetic restrictions, rest in bed, massage, or the wearing of an abdominal support, all of which will confirm the idea of disease in the patient's mind. It must be remembered that considerable variations in the chemistry, motility, shape and position of the stomach occur in normal individuals, and a departure from the average normal condition does not always indicate that this is the cause of a symptom, such as vomiting. When, therefore, the general evidence points to the vomiting being hysterical, psychotherapy should be attempted without any other treatment, even if a complete examination has been made and some slight abnormality has been discovered.

TREATMENT.

The treatment of hysterical vomiting has in the past generally consisted of isolation, rest in bed, dieting, administration of sedatives and, in some cases, a strict Weir Mitchell régime. In our earliest cases we occasionally used the stomach tube as a means of suggestion, but we soon dispensed with this, and during the last year and a half have used none of the methods mentioned, but have confined ourselves to treatment by explanation—the most rational form of psychotherapy. We sit alone in the room with the patient, after having examined him and come to the conclusion that he has no organic disease, and explain to him in language suited to his intelligence what we regard as the cause of his vomiting. We show him how, although at the onset it was the natural result of some irritation, disease or emotion, the primary cause is no longer present, and the persistence of the vomiting is simply due to the fact that the patient has formulated an idea that he is suffering from some disease of the stomach which necessitates his vomiting and consequently he continues to do so although it is no longer necessary. It is explained to him that his stomach is perfectly healthy, and if it is given sufficient work to do in the form of an ordinary food diet instead of nothing but slops, it will remain perfectly well, so long as the patient himself is confident that the explanation is correct. We do not leave him until we are satisfied that he is convinced of the truth of what we tell him. After this he is given an ordinary

full diet, and almost invariably he keeps this down, and a little encouragement on two or three subsequent occasions is all that is necessary to confirm the cure.

I believe the most important part of the treatment is its rapidity, and much better results can be obtained by attempting to cure the patient without any accessories in the way of isolation, diet, or drugs. The second essential for success is that the physician should be confident of his diagnosis and that the patient should have complete confidence in his physician.

17 Devonshire Street.

EARLY LESIONS IN THE GALL-BLADDER.

BY WM. CARPENTER MACCARTY, M. D., AND J. R. CORKERY, M. D.

The study of early pathologic conditions in the gall-bladder has been greatly facilitated by cholecystectomy, an operation which many surgeons have deemed advisable in preference to cholecystostomy.

In their experience, many cases in which the organ had been drained returned to them with their symptoms unrelieved. The desired relief in such cases seems to have been accomplished, at least in a much higher percentage, by the secondary complete removal of the organ. This experience with such cases following a secondary cholecystectomy has led, in the last five years, to the custom of primary cholecystectomy in preference to cholecystostomy in patients in whom there is a visible lesion and also in cases in whom there is no visible gross pathology but in which there is a definite clinical picture pointing to this organ plus enlargement of lymphatic glands along the ducts.

From January 1, 1913, to January 1, 1919, there were 4,998 gall-bladders removed. Of these, 4,824 (96.5 per cent.) showed unquestioned gross pathologic lesions according to the accompanying table.

In this series of conditions it may be seen that there were 157 with very slight lesions and 17 which were grossly "normal," most of which showed definite changes in the villi upon examination with a high power dissecting microscope or in microscopic sections.

CLASSIFICATION OF 4,998 GALL-BLADDERS.

	Specimens
I. Cholecystitis catarrhalis acuta,	17
Cholecystitis catarrhalis acuta (with "strawberry" appearance)	9
II. Cholecystitis catarrhalis subacuta	112
III. Cholecystitis catarrhalis chronica	2,021
Cholecystitis catarrhalis chronica (with "strawberry" appearance)	948
Cholecystitis catarrhalis chronica (with adenoma in the wall)	1
Cholecystitis catarrhalis chronica (with accessory fundus)	1

	Specimens
Cholecystitis catarrhalis chronica (with diverticula).....	4
Cholecystitis catarrhalis chronica (with old perforation) ..	1
Cholecystitis catarrhalis chronica (with very slight lesion)	38
Cholecystitis catarrhalis chronica (?) (3.1 per cent.).....	157
IV. Cholecystitis catarrhalis papillomatosa	212
Cholecystitis catarrhalis papillomatosa (with "strawberry" appearance)	129
Cholecystitis catarrhalis papillomatosa (with "strawberry" and cystic appearance).....	1
Cholecystitis catarrhalis papillomatosa (with a diverticu- lum)	1
Cholecystitis catarrhalis papillomatosa (with subacuta)...	1
Cholecystitis catarrhalis papillomatosa (malignum).....	1
Cholecystitis catarrhalis papillomatosa (malignum) (?)..	1
V. Cholecystitis catarrhalis carcinomatosa.....	22
Cholecystitis catarrhalis carcinomatosa (?).....	1
VI. Cholecystitis chronica.....	900
Cholecystitis chronica (with honeycomb appearance).....	8
Cholecystitis chronica (with perforation of wall).....	1
Cholecystitis chronica (with calcification of wall).....	1
VII. Cholecystitis chronica cystica.....	112
Cholecystitis chronica cystica (empyema).....	24
VIII. Cholecystitis acuta.....	81
Cholecystitis acuta (with perforation of wall).....	1
IX. Cholecystitis purulenta necrotica.....	168
Cholecystitis purulenta necrotica (with "strawberry" ap- pearance)	5
X. Cholecystitis ulcerosa.....	1
XI. Cholecystitis epitheliomatosa (with gall stones).....	1
XII. "Normal" gall-bladders (gross diagnosis) (.34 per cent.)..	17

The early changes in this organ consist of:—

1. Congestion and edema of the villi which are frequently associated with a bulbous appearance which, upon casual gross examination, makes the villi appear cystic. Occasionally they are cystic. The mucosa in advanced stages of this congestion and edema sometimes presents the appearance of being covered with small fish scales, an appearance which is due to the presence of a lipoid infiltration in the stroma or epithelial cells.

2. Local or general slight degree of leucocytic infiltration which manifests itself only in a slight enlargement of the villi and a cloudy or dull appearance.

3. Local or general slight degree of lymphocytic infiltration which if seen in the mucosa alone might possibly be considered

normal since the mucosa probably contains a certain number of lymphocytes, but when seen in association with a lymphocytic infiltration in the submucosa, muscularis and subserosa, is associated with a bulbous appearance of the villi or a thickening of the bases of the villi.

4. The presence of fibrosis in the villi which are usually not thin and tentacular (in sections) like those of the perfectly normal organ. The fibrosis sometimes extends into the submucosa, muscularis and subserosa.

5. The presence of lymphocytic infiltration and fibrosis such as is described above plus the presence of a finely granular or lipid substance in the epithelium or just below the epithelium in the mucosa.

6. The presence of slight or no lymphocytic infiltration and fibrosis plus the presence of large spheroidal cells filled with finely granular lipid substance in the mucosa and sometimes in the submucosa. These cells are similar to those which have been described in the so-called "strawberry" (1, 2) gall-bladder and in papillomata (3). This substance may not be visible grossly, but may sometimes be detected with the high power dissecting microscope. It is the substance which gives villi in the "strawberry" gall-bladder and papillomata their yellow or white appearance.

The conditions which have been described above do not alter the gross exterior of the organ, neither do they alter greatly the internal appearance to the naked eye.

It is this group of slight pathologic reactions which has made many surgeons, who believe in a cholecystectomy as the operation of choice in cholecystitis, somewhat slow in carrying out their belief in practice.

It is probably this group which is so frequently seen in association with stones and has led many observers to believe that stones occur in perfectly normal gall-bladders.

With our present knowledge we are not prepared to say definitely that such early conditions alone present sufficient symptoms to make a definite clinical symptom complex especially in view of the fact that recent studies made by the writer indicate that such conditions in the gall-bladder are also associated with somewhat similar changes in the extra- and intra-hepatic bile ducts, which might readily interfere with hepatic function and therefore pro-

duce general clinical disturbances. As a matter of fact, such patients do present some general disturbances which clinicians refer to under the broad heading of toxemia.

This paper has for its object the stimulation of greater interest and more detailed research in conditions of the bile passages which have heretofore been overlooked or thought to be normal.

DISCUSSION.

DR. HEMMETER: I thank Dr. MacCarty for his very interesting contribution compiled with so much diligence and such penetration. I cannot add to this very rare paper; but I would like to ask a few questions of Dr. MacCarty. Whether his biochemists at Rochester have made an analysis of these granular tissues that he has so often described beneath the epithelium. Dr. MacCarty calls it a lipoid substance. Is there any more exact chemical analysis that will permit us to say whether it is cholesterin? Or is it possible that propepsin is granular. We have, you know, in the salivary glands, and in the stomach glands, in the epithelium, certain granules which appear during rest and disappear again during work. They accumulate in rest. I would like to know whether that is a possibility here. Is there any relation of these beginning processes with any specific bacterial infection?

AN INTESTINAL TUBE.

BY MAX EINHORN, M. D.,

NEW YORK CITY.

At the first glance it appears hardly necessary to put any tubes in the intestine, after we have already the duodenal tube, which enables us to get the secretions which are most important for digestion right from the place of origin. It appeared to me, however, to be worth while to have some instrument by means of which we should be able to penetrate a little into the small intestine. It was constructed principally for radiographic purposes, in order to make visible the course of the small intestine, and I will show that it is possible to penetrate through the entire intestinal tract without harm to the individual.

The intestinal delineator was described by me in the *Medical Record*, and the apparatus is constructed in such a way that we can let it go farther into the small intestine and large bowel without the necessity of pulling it out. It has to have a length corresponding to the entire length of the digestive tract. The original instrument was twenty-nine feet long; but I think that probably a shorter one would be sufficient. I did not know, at first, how long it would need to be.

The instrument is swallowed by the patient, with a long string of copper wire, very flexible, so as to offer no resistance to the course of the intestine. If it was possible to let the instrument go through like that, and have the entire length of the intestinal tract filled by the instrument, and still have the patient able to eat, it also appeared feasible to have a small tube, quite long, that would do the same thing, and penetrate any depth we wished, without interfering with eating.

The intestinal tube that I have at present is about fifteen feet long. It is intended principally to penetrate the small intestine alone; but if we should want it to penetrate farther, the length of the tube would have to be increased five to eight feet more.

Now it is always difficult at first to arrange it so that there will be no harm done to the patient; and after we did the experiment, we gained the experience. The first patient that I gave the tube to was a young lady. She took it all right, and I warned

the house physician not to let it go in too fast. He did not heed the warning, with the result that the tube curled up in the stomach. That is the main difficulty in introducing the tube. You must wait, when a length of twenty-one or twenty-two inches has entered, which allows the tube to reach from the lips into the stomach and get to about the neighborhood of the pylorus. After a pause, we let it go in a little farther. After we have ascertained by fluoroscopic examination that it has reached the duodenum, we can let it go faster; but even then, it is not advisable to let it go down too fast; because at that time, swallowing pulls it in, and curling up in the stomach may occur.

I will let the tube go around, so that you can see its construction. Here is a little holder, which serves the purpose of helping to hold it in the mouth while the patient is asleep; so that the tube should be able to pass along, in case it is pulled farther in. There are also some markings here, made out of lead, and asphalt, and rounded, so that they are visible to the X-ray; but the tube is visible to the X-ray besides.

During eating, the tube should be fastened. After that, it can be let go. If these precautions are used, the tube gradually enters the small intestine, and is gradually pulled farther and farther, until it reaches the length it has to go.

I have a few X-ray pictures to show. The first will show the tube in the stomach in the faulty position, due to the faulty way of giving it. After that patient had had the tube in twenty-four hours, or so, I took a picture and found that it was curled up in the stomach. I took it out and gave the patient a day's rest, and afterwards repeated the experiment with these precautions.

(In answer to a question.) The instrument is made by Tieman and Company.

I can say a few more words. At the second experiment we let the tube go in gradually until it reached almost to the end of the small intestine. After it had remained there for three or four days, I pulled it back through the mouth. Now if we want to have a tube used for several patients, but only once in each case, we have it constructed so that there is a silk thread inside the tube; so that in case the pulling should break the tube and we should want to pull it out, the thread would hold it from going farther through. In the case of one patient in the hospital, this breaking of the tube took place. I pulled out seven feet

of it. I found that the tube was corroded by the products of digestion. I had used it in this patient several times, and then it broke; and seven or eight feet were left inside. This would not have worried me if the tube had been correctly situated in the small intestine; because the small intestine usually allows similar instruments to go only in a straight course. In the stomach and in the large bowel they act differently. In both of these, there is ample space for curling up; and the delineator comes out in a spiral formation. In the small intestine, however, the tube and similar instruments follow a straight course; that is, they follow the course of the small intestine.

At the time that I gave it to this patient, the X-ray apparatus was out of order and we could not follow the position of the instrument in that way. If we could have done so, we should have found out earlier that it was situated in the stomach and was curled up. When I found this out, it worried me; because if it is curled up and tangled, it is difficult for it to go through. But the patient felt well and made no complaint at all, so I thought I would wait and see. I gave no cathartic and let him eat the same food as usual; and after a few days, when the X-ray apparatus was in good shape, we found the remainder of the tube in the descending colon, curled up. The same evening we found the rest of the tube in the stool. That case has been related in order to show you what may happen. If, however, we are careful to see that it enters the duodenum first, before a great length goes into the stomach, there is no danger of such an occurrence as this; but after it does enter the duodenum, it is easy to cut through the end and let the tube pass through the intestines, as we do with the delineator. The only difficulty is if it curls in the stomach and does not enter the duodenum; but if it has entered the latter, which you can tell by the fluoroscope, there is no danger.

The intention of the tube is primarily a therapeutic one. If we can reach different parts of the intestine, we can easily see whether there is any affection there. When these are known to us, we can do something for their relief, if we have an instrument that reaches their location.

I will give you only one instance of the way in which it would be useful: that is, in severe ulcerative colitis of long standing, a case in which now, without this instrument, an appendicostomy

or colostomy would have to be done. By the use of this instrument, we can flush the bowels from above. We can wash the cecum as often as we want, leaving the instrument there; because the patient can eat without inconvenience.

I did that in one case. The patient was very sick and had chronic pancreatitis and jaundice. I gave him the tube, in order to see whether it would inconvenience him. It was important to ascertain the position of the duodenum. The X-ray man was in doubt as to whether there was an obstruction in the duodenum. This showed that the duodenum was free from obstruction. The tube passed in to the end of the small intestine. The patient ate as usual; and later, I pulled the tube out without harm. He was afterwards operated on, and cancer of the pancreas was found.

DISCUSSION.

DR. WILLY MEYER, New York: I just wish to make one remark with reference to the way that these patients with the duodenal or intestinal tube in can stand feeding. Usually the tube is employed for the purpose of duodenal alimentation. We have used it in cases of thrombo-angiitis obliterans for the flushing of the system. We believe that if we can get certain poisons out of the system, which we can do with the flushing, this seems to help the patients greatly; so for two years we have given the duodenal tube, allowing the tip to rest in the duodenum, and have flushed with nine or ten quarts of a solution, day after day.

Dr. Einhorn has used his intestinal tube with the intention of feeding his patients. We have used this tube for flushing. The patients take their meals for weeks and weeks. They are not annoyed by the presence of the tube. One would think that at the end of a long time they would be annoyed, but I have had patients take meals for eight weeks regularly, alongside of the tube without annoyance.

PRIMARY SARCOMA OF THE STOMACH WITH A REPORT OF A SUCCESSFULLY OPERATED CASE.

BY SEYMOUR BASCH, M. D.,

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Attending Physician to Lebanon Hospital.

NEW YORK CITY.

This subject has never before been brought before this Association for discussion. It is, however, of far greater clinical importance than is generally assumed. A careful study of the literature shows it to be of more frequent occurrence than individual experience would lead one to infer.

Relatively speaking, gastric sarcoma has more often been the subject of mistaken diagnosis than perhaps any other gastric condition. In many instances the clinical and operative diagnosis has been in doubt until cleared up by the ultimate histological examination. In such cases the presumptive diagnosis has almost invariably been cancer. Gastric sarcoma has, however, been mistaken also for many other intra- and extra-gastric conditions, *e. g.*, neoplasms of the spleen, tuberculous abdominal glands, abscess of the liver, suppurative peritonitis, pancreatic growths, benign peptic ulcer with secondary infiltrating tumor, etc.

The character of this neoplasm varies so widely in the different varieties from extreme malignancy down to almost certain promise of radical cure, that attention to the possibility of its occurrence, as well as to its early diagnosis, are matters of the utmost importance. In numerous instances the diagnosis has been made so late in the course of the disease that operative interference was tantamount to an antemortem procedure. Where, however, very early operation was undertaken, radical cure, or at least freedom from recurrence for quite a number of years, has frequently been obtained. It has been my fortune to have encountered such an early case, and I present it herewith in the hope of stimulating the interest of the members of this Association in this comparatively rare disease, and also of evoking an active discussion of the various phases of this subject.

The patient, C. C., aged 22 years, dressmaker, was first seen by me on January 1, 1919, in consultation with Dr. Joshua Leinir. The

family history, aside from the death of her father from gastric carcinoma, was negative. The patient herself, though a moderate eater, and somewhat constipated, had always been stout and enjoyed excellent health. She dated back her present illness to about one year before when, following a dietetic error, she suffered from what she termed an attack of acute indigestion. A laxative was followed by temporary relief for about three weeks when she began to experience frequent attacks of sharp, cutting pains in the right hypochondrium and anterior lumbar regions. These began several hours after meals, and lasted three or four hours each time. They were associated with heartburn and frequent belching of sour or tasteless gases. The distress was so acute that relief was sought through forced vomiting. The amounts vomited, especially of late, exceeded those ingested, although food from previous days was never noted. Meat and other "heavy" foods increased the pains, while fluids often gave relief. The pains, vomiting, etc., had markedly increased during the past three or four months, during which period, too, there had also been a loss of about twenty-five pounds in weight. There was no history of fever, cough, sweats, etc., or of hematemesis or melenae.

Examination showed a thin, anemic, and rather feeble individual, without, however, any evidence of systemic or central nervous disease. The abdominal wall was thin, soft and relaxed, the abdomen being readily palpable. In the right hypochondriacal and lumbar regions, somewhat to the right of the usual duodenal area, a hard globular mass, about three inches in diameter, could be very easily felt. It was smooth in outline, quite tender to the touch, and very freely movable. It was especially well felt when the patient arched her back forward, thus stretching the anterior abdominal wall. Its density and firmness reminded one forcibly of indurated tuberculous abdominal glands.

The stomach was considerably enlarged to percussion, the greater curvature being two or three inches below the umbilicus. There was no visible peristalsis or gastric stiffening, no resistance, no other points of tenderness, or any other palpable masses. The free edge of the liver was felt about two inches below the costal margin. It was, however, normal to the touch. The spleen and left kidney could not be felt, though the right kidney was prolapsed to the second degree. It was not sensitive to the touch and apparently normal.

For the purposes of better observation and treatment, the patient was admitted to Lebanon Hospital on January 3rd. Temperature, pulse and respiration were normal, the blood pressure averaged 105 systolic and 75 diastolic. Occasionally pains of moderate severity were felt in the right upper quadrant. She was placed upon a soft diet which was for the most part retained, although several times there were attacks of slight or even profuse vomiting, which, however, never was sanguineous, but always contained rather large amounts of mucus. The appetite was good. The Wassermann reaction was negative. Examination of both the urine and feces proved negative. Two

string tests were attempted, but proved unsuccessful as the patient vomited each time. Retention tests were also vomited, but one or two successful ones showed evidences of partial pyloric obstruction, viz., a few ounces of stomach contents, containing some of the barley and meat from the previous evening meal as well as a large number of sarcinae. There was no gastro-succorhea. In the fasting state the free hydrochloric acid was 10 and the total acidity 64; after the Ewald-Boas' test breakfast there was 30 free hydrochloric acid and 80 total acidity. Lactic acid was never present.

Diagnostic Considerations.—It was seen that we were dealing with an indurated tumor mass located in the middle area of the right side of the abdomen; this mass was definitely circumscribed, very easily palpable, freely movable, and rather tender to the touch. The tumor was causing a partial pyloric obstruction as well as pronounced abdominal pains, frequent attacks of vomiting and sitophobia, and was associated with a rapid loss in weight and strength. There was no evidence of gastro-intestinal hemorrhage.

Two pertinent points of interest presented themselves for discussion, viz., the nature of the tumor, and whether it was of intra- or extra-gastric origin. Although the patient was examined by a number of careful and experienced observers, no positive conclusion as to the nature of the tumor was reached. Neither the clinical course nor the objective findings offered sufficiently acceptable criteria for an indisputable diagnosis. The history of the case and the presence of an indurated tumor were strongly indicative of a malignant growth, and in an older individual, particularly with a longer history of gastric distress, the recent and rapidly progressive downward course, such a diagnosis would have been justified. But the age of the patient, the absence of free and of occult blood, the presence of sarcinae and of a high degree hydrochloric acid, were factors that spoke strongly against malignant disease, and favored rather the existence of a benign process. It was felt that we were dealing here most probably with a pyloric ulcer associated with pronounced inflammatory changes. Sarcoma was not even thought of. The tumor itself was very freely movable; its situation was very low for the pylorus, and the obstruction was only a partial and not a constant one. These facts led the greater number of observers to, therefore, favor the diagnosis of an extra-gastric lesion with inflammatory changes, resulting adhesions and partial

obstruction to the pyloric outlet. Amongst the other diagnoses suggested were omental tumor, inflamed adherent gall-bladder, tuberculous peritoneal glands, indurated inflammatory cyst, and even an unusually high situated chronic appendicitis. Discussion of the pros and cons for these various opinions is unnecessary.

The roentgenological examination pointed very decidedly to an intra-gastric lesion. The report stated that the stomach was enormously enlarged, markedly ptosed and atonic, with extremely sluggish peristalsis and a large residue after six and even after twenty-four hours. The duodenum was difficult to visualize, but the first portion and also the pyloric end of the stomach appeared decidedly pathological. The roentgenological diagnosis was "gastrectasy with marked pyloric obstruction, evidently due to an organic lesion involving the pyloric end of the stomach and the first portion of the duodenum."

In view of all the above mentioned facts the case was regarded as an unquestionably surgical one. Operative interference was advised and consented to. The operation was performed by Dr. Henry Roth on January 12th, ten days after the patient's admission to the hospital. A median line upper abdominal incision was made. The stomach was found to be very large and dilated and freely movable. The duodenum, too, was freely movable, but apparently normal. In the pyloric portion of the stomach there was seen and felt a large indurated mass, occupying the greater portion of the antrum and extending down very close to the pyloric ring. It was situated intramurally, the serous coat being intact. The tumor surface and outline were smooth, and while the mass strongly suggested an inflammatory infiltration, such as one so often finds associated with chronic ulcerative obstruction, Dr. Roth was decidedly of the opinion that it had the induration of a malignant process. Because of this fact, and also of the sharply circumscribed area and free mobility of the tumor mass a complete resection was decided upon. Thereupon, a typical Mayo (cautery) resection, which included the postpyloric portion of the duodenum and the stomach antrum just proximal to the tumor margin, was carried out. This procedure was followed by a typical posterior gastro-enterostomy without clamping. Very careful exploration of the entire abdomen failed to show the presence of any other growth. The wound was closed in the usual manner, leaving a rubber tissue drain in the duodenal stump.

The postoperative course during the first two weeks was marked by fever ranging from 102° to 104° F., and a disturbed mental state (psychosis) manifesting itself through extreme loquaciousness, irritability, distrustfulness of attending nurses and doctors, and a fixed stare with failure to reply to direct inquiries. On the thirteenth day after operation there was a sudden profuse discharge of a purulent secretion from the wound, followed by a rapid drop in the temperature, and a complete clearing up of the mental state.* Thereafter, recovery was very progressive, and the patient was discharged from the hospital on the twentieth postoperative day. She has steadily improved, and now, five months after her operation, feels very well and has gained over thirty pounds.

The specimen was microscopically examined, and reported upon as follows by the hospital pathologist, Dr. E. P. Bernstein.

A description of the microscopic picture presented by slide No. 16,178 is the following:

The mucous membrane directly over the main tumor mass is comparatively normal, showing only an occasional area of new growth cells between its ducts .

The submucosa which is the apparent origin of the growth, is almost completely replaced by tumor cells—either in masses or loosely distributed between strands of connective tissue. The cells are roughly polygonal in shape with a fairly large, well-staining nucleus and surrounded by an abundant zone of clear cytoplasm. Some cells show active mitotic figures. The tumor cells bear no relationship to the blood vessels present.

The muscularis is but sparsely invaded by tumor cells which are seen in the innermost portion of the circular layer but not at all in the longitudinal layer.

The serosa is normal.

The microscopic picture presented is that of a malignant tumor (large, round-celled sarcoma) which has not progressed enough to invade more than the submucosa.

In this case, as in so many others, practically all reported sarcomata of the stomach, the ultimate diagnosis was an unexpected surprise. Despite an increasing and ably discussed literature on the subject, this condition is still regarded as one of extreme rarity, so rare, indeed, that it is scarcely ever thought

*The rubber tissue drain had been removed rather early after the operation and this may have accounted for the retained exudate and the mental state.

of when confronted with a possible case. Writer after writer has, however, emphasized the fact that sarcoma of the stomach is a much more frequently occurring affection than is generally assumed, and has urged that more histological examinations be made of hastily assumed carcinomata.

In view of this fact, and also of the circumstances mentioned in the opening remarks of this presentation, it is felt that a brief discussion of the main clinico-pathological features will be of interest.*

Sarcoma may be either a primary or secondary growth of the stomach. Excepting in the case of the lymphosarcomata, the primary form is of more frequent occurrence.

Frequency.—From reports of cases it is generally assumed that sarcomata of the stomach constitute from 5 to 8 per cent. of primary malignant gastric neoplasms, and about 0.25 per cent. of sarcomata in general (Mikulicz and Kausch, Hosch). These and other statistical figures regarding this type of growth are relative only, since many cases of assumed cancer, or, on the other hand (as in the case now reported), of assumed chronic inflammatory infiltration, associated with chronic gastric ulcer, have upon subsequent histological examination, proved to be sarcomata. Thus, Perry and Shaw, in going over the Guy's Hospital series, found that four out of fifty previously reported "carcinomas" were really round-celled sarcomas.

Form and Size.—This growth may occur as small or large, nodular or diffuse hard flat tumor masses within the wall of the stomach, or as polypoid-shaped projections from the stomach wall into its lumen, or into the greater or lesser peritoneal cavity. The size varies from a minute nodule to an enormous mass that may as in Baldy's case weigh 15 lbs., and almost fill the entire abdominal cavity.

Tissue Origin and Types.—Sarcomata, being a non-epithelial type of growth, never originate from mucous membrane. They may, however, develop from connective tissue, smooth muscle fibre, lymphoid nodule, or from the endothelial cells of the lymph spaces of the stomach. According to their tissue origin they form respectively true fibrosarcomata, leiomyosarcomata (malignant

*Those desirous of obtaining further details of the subject will find them interestingly discussed in the writings of Schlesinger, Kundrat, Fenwick, Manges, Flebbe, Frazier, Warner, and others.

leiomyoblastomata), lymphosarcomata (malignant lymphoblastomata; Hodgkin's disease), and endotheliomata. According to their cellular structure they are classified as small and large round-celled and small and large spindle-celled sarcomata.

The Age of Incidence varies widely. Thus Findlayson reports a case observed in a child of three and one-half years of age, while di Giacoma one in a man of ninety-one years. Contrary to the accepted view regarding sarcomata in general, the age of greatest incidence appears to be not that of younger individuals, but that between 40 and 50 years (Corner and Fairbanks). The connective and lymphoid varieties occur especially in young individuals, while the smooth muscle type is seen more often after the age of thirty to fifty.

Situation.—While opinions differ, the majority of observers agree that the curvatures, especially the greater curvature, is the most frequent seat of origin.

Complications and Degenerations.—These are very numerous, and are associated with the more advanced stages of the growth. They include ulceration, hemorrhage, deformity of stomach outline, obstructions, torsion, cystic and purulent changes, adhesions to neighboring parts and metastasis. Metastasis may be entirely absent or may occur in neighboring or distant organs, particularly the skin.

1. *Symptoms.*—Frequently there is an entire absence of gastric symptoms throughout the entire course, or at least until the growth is far advanced. This arises from the fact that the mucous membrane is not involved and that there are no obstructive changes. In many other cases the predominant manifestations are those due to the complications, or to secondary changes in the growth itself, and these have given rise to greatest errors in diagnosis.

The gastric symptoms vary from the mildest expressions of gastric dyspepsia to the severest manifestations of gastric cancer. No definite diagnostic signs and symptoms can be stated. In almost all instances of sarcoma of the stomach, the correct diagnosis has never been made previous to direct exploration. In a few exceptions the diagnosis was ventured through the examination of tumor particles obtained from stomach contents (Riegel, Westphalen), or through excision of a metastatic growth in the rectum (Schlesinger), skin, glands, etc. That even this

latter procedure may be misleading, is emphasized by Leube, who instances a case of skin sarcomatosis occurring coincidentally with a true epithelial gastric cancer. Dreyer, on the other hand, encountered a case of spindle-celled pyloric tumor coincidental with a carcinomatous ulcer at the pylorus. The metastases in this case were carcinomatous. As long ago as 1902, Fenwick, in his well-known monograph on gastric tumors, cites a number of points, attention to which he claims makes possible the diagnosis of round-celled sarcoma of the stomach. Despite a careful search of the accumulated literature since that date, I have failed to encounter a single instance in which a correct pre-operative diagnosis has been reported excepting through the examination of a metastatic growth, or an expelled tumor fragment.

In a diseased condition, therefore, such as the one under consideration, the occurrence of which, even in a most typical manner, has almost invariably baffled the diagnostic acumen of so many careful observers, it would be an idle task to venture to lay down definitely drawn lines for exact diagnosis. Until the discovery of a specific reaction, the diagnosis will probably remain a matter of doubt. The one condition, after all, from which it is most desirable to differentiate sarcoma, is that of carcinoma of the stomach. The importance of this arises from the fact that sarcoma is, on the whole, a much more slowly growing tumor, and, in the relatively early cases, offers a far better chance for a radical cure. Both affections have many characteristics in common, such as the earmarks and accompaniments of malignancy, the alterations in gastric structure and functions, the occurrence of hemorrhages, metastases, splenic tumor, febrile states, etc. There are, however, definite distinguishing features which may serve to guide us in clinical differentiation. Thus, carcinoma arises from the epithelium, and though it does involve the rest of the stomach wall, the mucous membrane is always also affected. Sarcoma, on the contrary, as already stated, arises from the non-epithelial tissues, and does not as a rule invade the mucous membrane. When it does injure the mucosa, it does so through mechanical force (pressure, erosion, etc.). Carcinomata, too, are most frequently located at the orifices and in the body of the stomach; sarcomata along the curvatures. Hence, carcinomata are more apt to cause early pyloric obstruction. In most instances of sarcoma this is a late complication, and is

due not so much to narrowing of the pyloric orifice, as to massive infiltration of the stomach walls. In many instances this massive infiltration causes not an obstruction but a gaping of the pylorus. Carcinoma is very rapid in its growth, and in its general systemic effects; sarcoma shows quite the reverse characteristics, especially the leiomyosarcomata and the endotheliomata, which are very slow to metastasize. Hence, in carcinoma we more frequently find *early* local (gastric) symptoms and signs. The round-celled sarcoma, however, is, as a rule, a rapidly growing tumor, and, like carcinoma, very prone to metastases. Despite the fact that sarcoma frequently leaves the mucous membrane intact, a number of observers report diminished or absent hydrochloric acid, and even the presence of lactic acid and Boas-Oppler bacilli. Thus Schlesinger in three cases found free hydrochloric acid absent and lactic acid abundantly present; Mathien found hydrochloric acid absent in eleven out of seventeen cases. Indeed, Harlow Brooks reports that he found both lactic and hydrochloric acid present in his cases, and instanced this simultaneous presence of both kinds of acids as one of the signs that "should lead at least to the serious consideration of the possibility of gastric sarcoma."

Sarcomata being generally less malignant than carcinomata, we find that their average duration is longer than that of carcinomata. Two or three years' duration after the onset of symptoms is not rare.

Occult blood and hemorrhages are relatively less frequent in sarcoma than in carcinoma. Still, attention has been drawn by Manges to a group of sarcomata in which hematemesis is the leading symptom. These, for the most part, were advanced cases. Splenic tumor and fever are more frequently encountered in sarcoma than in cancer.

Finally, the spindle-celled sarcoma may progress to even quite an advanced stage without giving rise to any gastric symptoms. This applies more especially to the pediculated subperitoneal cases that project beyond the body of the stomach, invading the greater or lesser peritoneal cavities.

Prognosis.—The average duration of untreated round-celled types is reported to be about fifteen months; that of the spindle-celled twenty-four to thirty-two months, and that of the myosarcomata three and one-half years. The lymphosarcomata have

the greatest tendency to metastasize, and the endotheliomata the least. Naturally, the earlier the removal, the less danger is there of recurrence. Even the most prolific varieties—the lympho- and the fibrosarcomata—give a better prognosis as regards recurrence after removal than do carcinomata. In a case of gastric sarcoma reported by Ruppert the patient was alive and free from recurrences fourteen and one-half years after operation.

Treatment.—This, of course, in cases not too far advanced, or even in these, where the diagnosis is in doubt or mechanical relief is indicated, should only be surgical. In inoperable cases a course of Coley's "serum" might be tried. In cases of lympho-sarcoma, especially in the inflammatory or Hodgkin's type, arsenic therapy is decidedly indicated.

40 W. 88th Street.

DISCUSSIONS.

DR. LOUIS M. GOMPERTZ, New Haven, Conn.: Like Dr. Basch, I want to report a case of sarcoma of the stomach in which a mistaken diagnosis was made. On March 25, 1908, a man forty years of age, consulted me for digestive disturbances, which had existed for about two years, but had become aggravated during the last few months. Pressure and fullness coming on soon after eating and vomiting about two hours after meals were the most prominent symptoms. Pain was entirely absent. There was a loss of about 10 pounds in weight during the last two months. The physical examination of the patient disclosed nothing of importance. The microscopical examination of the fasting stomach contents after a test supper gave evidence of retention. Food remnants and lactic acid bacilli were found. The chemical examination after a test breakfast showed an absence of HCl and the gastric ferments, with the presence of lactic acid and blood. A diagnosis of carcinoma of the pylorus was made.

He was operated upon by Dr. Wm. F. Verdi, of New Haven, on April 12, 1908. A pylorectomy and posterior gastro-enterostomy was performed.

The removed growth was examined at the pathological laboratory at Yale Medical School and found to be a spindle-celled sarcoma.

The patient was examined by me one week ago. The fluoroscope and X-ray gave negative results. The microscopical examination of his fasting stomach contents gave no evidence of retention. After a test breakfast there was an absence of HCl and the gastric ferments. He had gained 15 pounds since the operation and is in perfect health today.

DR. DUDLEY ROBERTS, New York: In all probability the case that Dr. Basch reports was a sarcoma of the stomach, as he states, but

I think that one should be very careful in making a pathological diagnosis of sarcoma of the stomach. Such a report was made on an inoperable tumor in a case under my observation some years ago, in spite of a positive luetic history and microscopic appearance of the tumor, which I would now regard as being a gummatous lesion. Syphilis of the stomach is by no means as rare as we have believed, and I am convinced many supposed carcinomas and some of the reported sarcomas are of syphilitic origin.

DR. SEYMOUR BASCH, New York (closing): Dr. Gompertz has brought up the interesting point of the change in the secretion in these types of growths, and the fact that in the majority of cases the mucous membrane is uninvolved has been referred to by Dr. Louria. To properly discuss these phases would bring up the whole question of achylia. We know that many cases of chronic achylia have histologically intact cells of the secreting glands. On the other hand, in the early stages of carcinoma, there is an infiltrating gastritis, which produces a diminution in the gastric secretion. Years ago, Brooks, who had observed four cases of sarcoma of the stomach, was struck by the fact that both hydrochloric and lactic acid were present in these cases. He considered it a diagnostic point. The fact that Dr. Gompertz's case showed lactic acid, points to an infiltrating gastritis or to stagnation of gastric contents either prior to or coincident with the development of sarcoma.

In classifying sarcoma one cannot go by the cell variety, but has to know the tissue origin; because there is a transition of round-celled to spindle-celled sarcoma, and so on.

Regarding Dr. Louria's case, I think that it might have been a secondary sarcoma. Sarcoma is not always primary, even when you do not see a tumor elsewhere. In the lymphatic type, you may have a small nodule somewhere else in the body and overlook it.

I could not go into all the details in my paper, but cases have been described that have filled the abdomen, weighing, in some cases, as much as twelve pounds, without producing stomach obstruction. It is particularly the polypoid form which grows outward and invades the peritoneal cavity that does not produce gastric obstruction.

Regarding lues this question was taken up by the pathologist, Dr. Bernstein, and myself. Yet I question whether any luetic affection would invade the submucosa and no other part of the stomach wall. Gummas will invade the tissues. The pathological report showed large round cells, and the submucosa only was affected. A Wassermann was taken, and this should always be done, particularly in the case of young persons with tumors of the stomach.

DIAGNOSIS OF DISEASES OF THE PANCREAS.

BY THOMAS R. BROWN, M. D.

There is probably no organ of such importance in the human body the organic and functional diseases of which are less frequently and less correctly diagnosed than the pancreas.

In regard to its internal secretion, closely associated with the islands of Langerhans, and its rôle in the metabolism of the carbohydrates, a fairly clear conception is held, but the study of its external secretions in health and in disease is so difficult and so complex that the aid derived therefrom is comparatively slight in the diagnosis of pancreatic disease, although it is highly probable that many, if not most, of the organic diseases of this organ are associated with variations—quantitative or qualitative—in these secretions, and it seems obvious that an organ with such a large and varied secretory function and complex nervous supply, and one of such fundamental importance in the elaboration of practically all types of foodstuffs, must show marked functional disturbances could we but unravel them. It seems, therefore, wise in this paper to first sketch very briefly the salient points in the anatomy and physiology of this organ, then to take up seriatim the various means of diagnosis at our command, and last of all to review the various diseases of the pancreas in the light of these diagnostic methods, and, with a real knowledge of the organ's physiology, see if by the help of the newer modes of study we can get a step nearer to the correct diagnosis of its various pathological entities.

The names of some of the greatest physiologists and anatomists are linked with our knowledge of the anatomy and physiology of the pancreas—Claude Bernard studied the rôle of the pancreatic enzymes in the digestion of fats, proteids and starches, Heidenhain demonstrated the gross and microscopic changes in this organ during digestion, Langerhans described the islands bearing his name, and which subsequently were shown to be of such paramount importance in the combustion of the sugars; in recent years our knowledge has been immensely enriched by the work of Pawlow and his pupils and Bayliss and Starling, while with the clinical and surgical sides the names of Friedreich, Fitz, Oser,

Moynihan, Mayo-Robson, Von Mikulicz, Bozeman and Gussenbauer are closely allied.

In regard to the *anatomy* of the pancreas, it is well to remember that it is subject to great variations in size; that it is very difficult to palpate through the abdominal wall, and, in fact, even when the abdomen is opened surgically; that its head lies in the concavity of the duodenum, and that head, body or tail lies in close juxtaposition to stomach, liver, gall-bladder or transverse colon as well; that it is subject to anomalies such as annular pancreas or accessory glands which are not uncommon—which may be in the gastric or duodenal wall, or below the pancreas even as far down as the ileum.

The character of the ducts, however, is by far the most important point, anatomically considered, which we must remember relative to the etiology and diagnosis of pancreatic disease; the lower that of Wirsung larger in about four-fifths of the cases; the upper, that of Santorini, usually anastomosing with the former and larger in *about* only one-fifth of the cases. In the vast majority of cases the common duct, which in three-eighths of the cases lies in a groove on the surface of the gland, and in five-eighths passes through the head of the pancreas, joins with the duct of Wirsung to form the diverticulum of Vater—a short conical cavity represented in the duodenum by the papilla of Vater—recognized by a small elevation of the duodenal mucous membrane—and it is this, the usual though not the universal formation (for occasionally common duct and duct of Wirsung open separately) which is the fundamental cause of the intimate connection between pancreatic and gall-bladder disease, especially in cases of stone in common duct or in diverticulum of Vater.

In regard to the *physiology* of the pancreas it is only necessary to mention the fundamental rôle played by secretin in calling forth the secretion of the gland derived from the duodenal pro-secretin by the action of acids, though peptones, salt, lipoids, fats and soaps can also activate the pancreas. We believe with Pawlow, however, that there is a nervous, as well as a hormonal control of pancreatic secretion, and we feel that our quantitative ferment studies of the pancreatic secretions in achylia gastrica strongly support this view—a view which Ehrmann also holds from somewhat similar studies using a different method. As regards the pancreatic ferments called forth by either mechan-

ism, all, of course, admit the presence of diastase or amylopsin, lipase or steapsin, and trypsin-zymogen or trypsinogen to be subsequently activated by the duodenal enterokinase, but there is still much discussion as to the possible presence of other ferments or pro-ferments such as rennin, a hemolysin-zymogen, nuclease and a lactase in children. We have shown by dog-experiments the primary stimulating and secondary inhibiting effect of bile upon pancreatic secretion, of considerable clinical importance in the case of pancreatic disease associated with jaundice, while Wohlgemuth lays especial stress upon the opposing effect of adrenal and pancreas, and Eppinger, Falta and Rudinger the intimate relationship between thyroid, adrenal and pancreas, of peculiar interest in connection with the study of the internal pancreatic secretion, apparently originating in the islands of Langerhans.

Disturbance of this internal secretion, either spontaneous or as an alimentary glycosuria after the administration of a large sugar meal, may be of considerable help in the diagnosis of diseases of the pancreas, although it is very striking how extensive changes may occur in the islands of Langerhans with no disturbance of the external secretion and vice versa.

In discussing the means of diagnosing pancreatic diseases, let us in the first place call attention to the fact that an absolute diagnosis is possible only in a comparatively few cases, such as certain examples of pancreatic cyst and certain types of carcinoma of the head of the pancreas, and possibly acute pancreatitis; in most cases the diagnosis must be only presumptive; in the second place to note that, however definite the evidences that we are able to demonstrate of functional disturbances, these furnish no proof of organic disease of this organ, although combined with other signs and symptoms these variations may be of inestimable value.

Let us take up in order the functional diagnosis, the etiological factors involved, and the signs and symptoms derived from the usual methods employed in studying disease, following these with a few words as to their application in the diagnosis of the various recognized pathological lesions.

The *functional diagnosis* may have to do with the study of the external secretion or of the internal secretion—the former, in our experience, of far more significance in the vast majority of cases.

As regards metabolic studies, determinations of the unabsorbed protein, fat and carbohydrates are all of value, though obviously of far less significance if associated with diarrhea with its marked diminution of the time of the transit of the foodstuffs through the intestinal tract. An *azotorrhea* is certainly not so significant as the corresponding disturbances of fat absorption due to the other accessory mechanisms for the digestion of the proteins, the pepsin and hydrochloric acid and erepsin, for example, and the definitely noted fact that even with marked disease of the pancreas there may be very little waste of nitrogen.

The finding of bits of striped muscle or of thymus or other tissue rich in nuclei is, however, of more significance than data obtained from the pure metabolic studies of nitrogen loss, especially if associated with a fatty stool and in the absence of diarrhea, as of all the proteins these, perhaps, are more in need of pancreatic juice for complete digestion, although few agree with Schmidt's dictum that the nuclease of the pancreas is the sole ferment capable of digesting nuclei, most believing that they can be digested by stomach and intestinal juices, or even possibly by the ferments from bacteria without the aid of the pancreatic secretion; in other words, nuclear digestion is *not* due to a specific nuclease.

In our experience the various older methods of showing deficient tryptic digestion, such as the glutoid capsule or gelodurat capsule test are open to the same criticism; while direct methods of demonstrating trypsin quantitatively in the stool by the use of serum agar plates or by the casein method are not of great value again because erepsin or bacterial ferments may produce almost the same reactions; and the attempt at studying the gastric contents after an oil meal with its subsequent effect in producing a regurgitation of duodenal contents is open to considerable technical difficulty. Recently, however, the study of the duodenal contents for all three ferments, obtained by direct aspiration through an Einhorn or similar tube is of unquestionable qualitative though more doubtful quantitative value.

Unquestionably data of great value may be derived from the study of the fat in the stools, especially in the absence of jaundice. Whether shown as a fatty diarrhea or a butter stool, whether by metabolic studies we find that 50 to 70 per cent. of the fat is not absorbed or whether by chemical investigation we

show that of this unused fat 50 to 60 per cent. is in the form of neutral fat, possibly with an associated increase in lecithin, it is an undoubted fact that such marked disturbances of fat digestion—macroscopic, microscopic or chemical are of extreme significance in the diagnosis of pancreatic disease.

As regards the other important ferment of the pancreas, diastase, we have always felt that it offered a singularly attractive field for study in this connection. In the first place if we eliminate the salivary diastase, a very simple problem technically, the only other sources of this ferment are Brunner's glands and possibly the liver where, however, they are present in almost negligible amounts; in the second place the myriad ferments met with in the small intestine have very little influence on this ferment, and in the third place diastase is so much more stable than lipase that it lends itself peculiarly well to *quantitative* determination, in our opinion of fundamental importance in pancreatic diagnosis.

Of course, many methods have been suggested, but it has seemed to us that most of these were not sufficiently rigorous as regards technique to make their figures absolutely reliable.

By a method described by us a few years ago by which we believe that we have reduced error to a minimum, we determined that the readings of fifteen normal cases varied from 600 to 2400 units, and as we firmly believe in a quantitative as well as a qualitative response on the part of the pancreas to a definite food-stimulus, we feel that these figures furnish a valuable criterion. At the same time we determined the normal amount of the diastase in the urine in 44 cases, and here we found the limits from 15 to 120 units, but in our experience, except for the enormous increase in pancreatic calculus, of which we have seen one case and studied the urine, the estimation of the diastase in the urine is of more value in determining renal function than as an aid in the diagnosis of pancreatic disease. Certainly in our opinion, the quantitative estimation of the diastase in the stool is the easiest technically, has the most clean-cut end-reaction and is less open to criticism than any other method attempting to quantitatively estimate pancreatic function.

The various tests for the internal secretion of the pancreas, the adrenalin mydriasis, the permanent or transitory appearance of sugar in the urine, and the alimentary glycosuria test may

furnish data of interest and value, although, as we have said before, there is a singular lack of co-ordination between glycolytic function and extent of damage to the purely glandular portion of the pancreas. In our experience the Cammidge reading is found in far too many other diseases and is too often absent in extensive pancreatic lesions to be of value.

And now a word about *etiological* factors before discussing general and special symptoms, important because in many cases of pancreatic disease, the history of certain factors in the patient's past history or the finding of certain diseases elsewhere furnish us with data of real value in suggesting possible secondary pancreatic involvement. The etiological factors, of varying degrees of importance, are catarrhal inflammation of stomach and intestines with its possibility of ascending infection of the pancreatic ducts by various bacteria, notably the *Bacillus coli* and certain cocci, or by the products of decomposition or inflammation, and, of ever greater import, (catarrhal) inflammation of gall-bladder or gall ducts, or gall-stones—these two, especially disease of the biliary system, being by far the most important etiological factors—easily understood if we remember the anatomy of biliary and pancreatic ducts. Other etiological factors, all of considerably less importance, are trauma, which possibly plays a rôle in the so-called pancreatic apoplexy and in cysts of the pancreas; arteriosclerosis, of especial interest in the development of the various cirrhotic conditions; syphilis, in which gumma of the pancreas may be found though it apparently plays a very slight rôle in chronic pancreatitis; alcohol, a possible factor in pancreatitis, and obviously various inflammations or neoplasms of the contiguous organs which may involve the pancreas by extension or by metastasis.

As regards the *symptoms* of pancreatic disease, they are, as a rule, very vague, very blurred, as in the main they are those common to all digestive diseases if chronic, such as fullness, pressure, gas, pyrosis, sometimes nausea and vomiting, loss of appetite, etc., while they practically always simulate those of certain other acute abdominal conditions, if acute. Certain symptoms should be especially mentioned—*sialorrhœa* is often considered an important symptom, but in our experience is more frequently met with in diseases of the duodenum; *pain* is somewhat suggestive when present, as it is often uncommonly violent,

sudden and colicky, and it has been the first striking symptom in a few of our cases of carcinoma, sometimes associated with local sensitiveness; *constipation* is the rule, though diarrhea is sometimes found; *loss of weight* and *strength* is often a striking symptom especially in comparison with the paucity of other findings, and, of course, is peculiarly striking in the obese in whom pancreatic disease is more common; *jaundice* if present is very important, and a persistent, usually in the main, painless jaundice in persons who have passed middle life is very suggestive of carcinoma of the head of the pancreas, or much less frequently of cirrhosis of the pancreas; a palpable *tumor* that is definitely shown to be pancreatic is very difficult to demonstrate, because of the consistency of the pancreas and the character of the overlying organs, and is rarely determined in abscess or carcinoma, much oftener in cyst, its situation is usually median, just above the umbilicus, occasionally to right or left, and its location may sometimes be determined better by inflation of stomach or colon; a diffuse shotty condition of the body fat, especially that of the abdomen, has seemed to Dr. Finney and to myself as somewhat significant of pancreatic disease as verified by subsequent operation.

These signs and symptoms, in the main not clear-cut, are, with the functional tests already mentioned, and possibly with the aid of other laboratory tests, such as the lessening of indican or of ethereal sulphates or the presence of fat or maltose in the urine, our only means of making a diagnosis, and thus, obviously even with considerable experience, medical and surgical, correct diagnosis is the exception rather than the rule, with, of course, certain exceptions, notably certain types of pancreatic cancer, cyst, and possibly acute hemorrhagic pancreatitis. The picture of *acute pancreatitis* with its three stages, the hemorrhagic, gangrenous and suppurative, is not one of infection or inflammation, but one of toxemia or shock, and high intestinal obstruction, and with its profound prostration, thready pulse, lack of fever, and absence of leucocytosis, sensitive epigastrium without muscle spasm, and the frequent previous history of gall-bladder or duodenal disease, especially the former, should make us suspect this condition more frequently than we usually do, while in cases with this history, not ending fatally and not operated upon, we should always be on the lookout for the symptoms

of abscess or gangreen as late sequelae. At operation, of course, the finding of evidences of fat necrosis is of extreme significance. As regards *pancreatic calculus*, a very rare condition, the symptoms are suggestive of biliary colic without jaundice, but if careful stool and urine studies are made a marked lessening of diastase in the former and enormous increase in the latter can be noted, as we have done in one case. Here as in cysts the X-ray may be helpful.

In cases of *chronic pancreatitis* or *cirrhosis of the pancreas*, the history of the usual etiological factors, the vagueness of the symptoms with their intractability to the usual methods of treatment, the increasing weakness of the patient without demonstrable cause, sometimes the deficiency of digestion of the various foodstuffs, notably the fats, as shown by stool studies after a Schmidt diet, should make us suspicious, and in our experience a quantitative stool study often demonstrates not the absence but a marked diminution of the diastase, as, for example, in six cases so studied, our figures read from 33 to 120 units in comparison with our normal readings of from 600 to 2400 units. In cases of chronic pancreatitis associated with jaundice, the differentiation from carcinomas is often very difficult. In cirrhosis of the pancreas, alimentary glycosuria is common, as might be suspected.

Cancer of the pancreas is usually of the scirrhus type, most often confined to the head of the organ, and if associated with jaundice produces a rather characteristic picture—usually progressive jaundice often tending to a mahogany brown color of the patient, with loss of weight and strength, often fatty stool, often alimentary glycosuria, usually distended gall-bladder—a point of real importance in differentiating it from obstructive jaundice due to gall-stone, often a previous history of gall-bladder disease, while in our experience there is in almost all cases a practically complete disappearance of diastase from the stool—in five cases there being always less than twelve units, below which we have not tested as we regard this as the limit of error.

In the case of the types of pancreatic carcinoma without jaundice, the latent and those with marked loss of weight and strength, with no apparent cause, we can only suspect the diagnosis by the course of the disease and its lack of response to any treatment, and stool studies, especially quantitative estimations of

the ferment should prove helpful. In our experience the only other condition showing a complete absence of the pancreatic ferment in the stool is sprue and here the differential diagnosis should not be difficult.

We have seen a few cases of pancreatic carcinoma in which the diastase is not absent, but is present in reduced or even in normal amounts, but the complete absence of diastatic ferment is the more probable finding. Thus, while presence of diastatic ferment does not rule out carcinoma, absence of diastatic ferment is extremely significant, in our opinion, in making this diagnosis.

Tuberculosis and *syphilis* of the pancreas are comparatively rare and offer no characteristic symptom-complexes; in the case of visceral lues gummata are occasionally found in the pancreas, but whether syphilis plays an important rôle in the production of diffuse indurative or cirrhotic processes is open to much doubt. *Fat necrosis* is of singular interest from the pathological standpoint as an evidence of the effect of aberrant pancreatic secretion upon the fat of the body, notably, of course, that of the abdomen in the neighborhood of the pancreas; clinically its especial significance is, if found at operation, to suggest the presence of pancreatic disease, especially the various forms of pancreatitis.

Cysts of the *pancreas* have obviously been given special attention, diagnostically speaking, because of their greater ease of diagnosis and their obvious surgical therapy; local pain of a colicky nature, a round or oval mass usually in the middle line about the umbilicus, small, sometimes fluctuating, more often tense, with occasionally slight and rarely marked spontaneous changes in size, sometimes if situated in the head of the organ (a rather uncommon finding) associated with jaundice, occasionally diagnosticated by puncture and finding the ferments or preferments, are the points of especial diagnostic interest.

As to *functional disturbances*, we have always felt that an organ of such complexity must show marked functional variation due to nervous and other influences, but at the present writing no definite facts are available. Gross believed that the diarrhea met with in achylia gastrica was due to a functional pancreatic achylia, but our and other quantitative stool studies disproved this, at least in our series of cases. Nevertheless, we

feel that this offers a very fertile field for future study, and we are now carrying on certain investigations along these lines.

This, I take it, is practically the present status of our knowledge as regards diagnosis of diseases of the pancreas—not a very satisfactory showing if we are to judge by the percentage of correct diagnoses made of diseases of this organ as subsequently demonstrated by the results of surgery or by the findings at autopsy—but yet so pregnant with possibilities, so crammed full of suggestive fields of intensive study, mainly, of course, along more intensive clinical lines on the one hand, and, on the other, chemical, serological or enzymic routes that I feel sure that within the next decade by the improvement of the methods described in this paper, and by the elaboration of newer, more accurate and more refined means of study, we should make enormous strides in the diagnosis of both functional and organic diseases of this organ.

DISCUSSIONS.

DR. BURRILL B. CROHN, New York: It was a great privilege and pleasure to listen to this paper. The whole subject of the diagnosis of pancreatic disease is one on which there has been a great deal written and for which a great many tests have been devised. The tests in the literature do not stand close scientific analysis or hold water when investigated. For this reason, it is a pleasure to hear Dr. Brown express his opinions, which are very conservative, because he appreciates the difficulty of making straightforward or clear-cut statements.

Dr. Brown has chosen amylase as the most constant ferment in the pancreas, and has used it by testing the amylase reaction in the stools. I prefer to get the ferments directly from the duodenum by means of the tube, thinking that they will not then be contaminated by the secretions in the intestinal tract. I was a little shy of the amylase reaction, which I found could be markedly influenced by serum. If a fraction of the drop of serum only were present, it would enormously intensify the amylase reaction. In trying to find out which constituent of the serum it was that intensified it, we dialyzed the serum and found that it was the mineral salts. This made us careful of working with amylase. I personally rely more on trypsin, which is a more stable ferment.

The question of shock is interesting. I had a case of acute pancreatitis in which the shock was enormous. The woman lived for five days after the operation, and was mentally clear almost until her death; yet she had an almost impalpable pulse, with cold, clammy extremities. Dr. Evans denied that it was shock, and said that it

was toxemia. I could not agree with him. The surgeon, at the operation palpated the pancreas and said that he thought that it was normal; but he stated that he agreed with Deaver that no one had a right to express such an opinion, even at operation. If, however, we accepted that dictum, we should never be able to make a diagnosis at operation. One would think that the surgeon should have known that there was something the matter with the pancreas when he reported it as normal; for within two days the patient was exuding pancreatic secretion, which rapidly eroded the abdominal wall. As Dr. Brown says, the disease can be diagnosed early by the ferments in the stool and in the duodenal contents. The ferments are very hard to estimate. Quantitative determinations are difficult to make, but the demonstration of diminished ferments in the pancreatic secretion long precedes the appearance in the stools of neutral fat or the increase of undigested muscle fibers. I have noted two cases with sclerosis of the pancreas and stenosis of the ducts at the head of the pancreas, in which steatorrhoea and creatorrhoea appeared long before there was obstruction of the ducts. One of these cases came to autopsy, and finding of patent ducts was confirmed by the pathologist at that time.

I was sorry to hear that Dr. Brown had discovered achylia in sprue. There is a case going home from the hospital today in which Dr. Libman and myself thought that we were the first to suspect that there is achylia in sprue. That brings up the question of achylia pancreatica and whether it can be explained on the same basis as achylia gastrica. It does not mean entire absence of the secretion of the pancreas. You may have complete absence of pancreatic secretion from obstruction, and yet have no marked disturbance of fat and nitrogen absorption; and you may have the reverse of this. The two do not depend on each other.

DR. MAX EINHORN, New York: I have to say only a few words. I cannot agree with Dr. Brown. It is quite clear that if you want to study pancreatic disease, you should study the pancreatic juice in its purest form. Just as you take the gastric juice from the stomach, not waiting for it to pass through the intestinal tract, you should take the pancreatic juice from the spot that it comes from. Whether you accomplish something or not, that is not the fault of the method. You should try, at least, to see what you can do. There are a great many organs that are diseased, and not all of them show a diminution in their functional activity. There are diseases in which the function is hyperactive. You have that in the stomach. In the same way, in disease of the pancreas, we should not only look for an absence of the ferments, but also try to see whether the function is not increased. I have convinced myself that there are conditions in which pancreatic activity is really increased. I have written on the subject several times, and have worked out a method by which we can determine all three ferments. If you take all, I

think you can draw more conclusions from your investigation than if you merely took one. If we can have all three ferments, why not do so? Why not see how they stand in relation to one another? We can do that by using the agar tubes, the way I devised them; but you must see that they are good, that they are not too old, and you must always work in the same way. Then you can reach conclusions.

Dr. Brown maintains that in achylia gastrica all the ferments are there. I have studied them, and have found that in achylia gastrica we do find cases in which the pancreatic juice is abnormal, some parts of it being increased, and others diminished. Again, in pancreatic cancer, I have shown repeatedly that in cases that come to operation, in which we could verify the finding, the trypsin ferment was absent entirely already, before the operation. I have studied these cases and found amyllopsin present, steapsin present but diminished, and trypsin absent.

In regard to sprue, I think that not all cases are troubled with achylia. In many it is present, but in some there is subacidity, and in some normal gastric secretion. It is the same with regard to the pancreas.

DR. B. B. VINCENT LYON, Philadelphia: This very excellent paper of Dr. Brown's has opened up many angles of profitable discussion.

I desire to emphasize just one point in the etiology of pancreatitis. In addition to the other etiological factors Dr. Brown mentioned he brought out the factor of ascending infection of the pancreatic duct due to swelling of the duodenal mucosa associated with and following gastro-duodenal catarrh—presumably of infective origin. In such cases I believe we must look higher than the gastro-duodenal zones if we are to find the primary breeding grounds of the infective agents that eventually lead to ascending infection of the pancreatic or of the common bile duct. I believe that if we direct our attention aborally that we will find more cases of pancreatitis and of gall duct and bladder disease to derive the primary etiological factor from infections of the gums, teeth, tonsils, nasal sinuses and of the bronchial tree.

The swallowing of infected saliva inaugurates a successful implantation of the infection in the gastric mucosa provided the mucosa in the individual case is receptive to infection. The infective agents are carried through the stomach with the passage of chyme or in concentrated doses by the peristaltic passage of the fasting gastric residuum to subsequently create a catarrhal duodenitis, infective or otherwise, as determined by the individual patient's local resistance. Once the duodenum is infected it is a very easy, and by no means infrequent step to ascending infection of the terminal portion of the pancreatic and common bile ducts.

To illustrate: One of the officers serving with my unit abroad, previously in good health, suffered a rather severe attack of influenza.

Later he developed an infective gastritis, and shortly afterwards an infective duodenitis. Before many weeks had gone by he developed a true diabetes. Was this the result of an ascending infection of the pancreatic duct, with sufficient cloudy swelling of the duct to block off pancreatic secretion, to produce intra-pancreatic pressure effects, perhaps, involving the Islands of Langerhans? Who really can say? Is such a theory tenable? At any rate, studies in his case by duodenal intubage showed that he had an infected duodenitis and cholelchitis with cultural recovery of bacterial groups that were identical with those most commonly encountered in our epidemic influenza cases.

I agree with Dr. Crohn and with Dr. Einhorn that our best instrument in the diagnosis of pancreatitis and of diseases of the duodenal zone is the duodenal tube. But it must be scientifically used. By this I mean that the material recovered from the duodenal zone must not be casually inspected and thrown away, but should be carefully studied cytologically, bacteriologically and micro-chemically. Perhaps some of the missing links in our newer conceptions of pancreatitis and diseases of the biliary tract will thereby be supplied.

It is needless to say that we should not omit our stool and other associated studies in pancreatic diagnosis.

DR. W. C. ALVAREZ, San Francisco: I certainly wish Dr. Brown well in his work, for I have long wished that we could diagnose a failing pancreatic function. Seven or eight years ago when the Wohlgemuth method was popular, I did six hundred tests, one after the other on the patients as they came in. I soon discovered that the amounts of diastase found depended partly on the rate with which the fecal material had come through the colon. The constipated stools had little diastase. In practically all of the cases studied we found considerable amounts of diastase and trypsin. About the only ones in whom the secretion was found absent were three cases of carcinoma of the pancreas. In one of these, operation showed that the organ was almost replaced by the tumor. Yet on certain days we could find practically normal amounts of diastase in his stools. This discouraged me so much that I gave up the test and have seldom used it since. It was plain that I could not hope to detect moderate grades of pancreatic insufficiency, if such exist. Another puzzling thing was that some stools which were full of diastase showed a great deal of undigested starch, while others with a low diastase showed very little starch. Why this should be I do not know.

DR. EMMANUEL LIBMAN, New York: I just want to discuss two points. In the first place, the question of the diagnosis of acute pancreatitis. This is not so difficult, if one realizes that there are symptoms that develop from the extension of the inflammatory process in the left mesocolon, giving pain and tenderness in the left lumbar region. That is present, even in people who are not sensitive to pain.

I want to speak, also of achylia in sprue. Several years ago, a paper was read by Dr. Wood, of Wilmington. I asked him whether

studies had been made of the gastric function in sprue, and he replied that he did not know of any. He suspected that achylia might be present. Since then, Dr. Wood and others have made observations to show that in sprue there is often an achylia.

I had an opportunity of seeing one case of sprue recently, and one case of secondary sprue; and I thought that the probability was that there would be a diminution or absence of pancreatic ferments. I have just had a case come from the West Indies, and want to study it to see whether there is not achylia. Dr. Brown's studies are most important. It is now necessary to confirm them by means of studies of the duodenal contents.

DR. W. A. BASTEDO, New York: When Dr. Brown, several years ago, reported a case of achylia pancreatica in sprue before this association, I also reported a case. In my case there was a high hydrochloric acidity in the stomach. At the same time there were canker sores in the mouth and erosions of similar character in the rectum, and also the symptoms of ulcer of the stomach. I assumed, because it was irritable, that there were erosions in the stomach, and that these accounted for the high acidity.

DR. THOMAS H. BROWN, Baltimore: I wish, in closing the discussion on my paper, to make clear to you two points: I have studied both the stool and the duodenal contents. While the former is less desirable in some respects, it saves more for quantitative estimations. It is very hard for me to understand how you can measure the entire secretion of the pancreas by duodenal aspiration, or are able to give a well-balanced stimulating meal without clogging up the tube. Both methods should, of course, be employed together. Naturally, we all recognize that there are exceptions to all rules. We have had cases of carcinoma with pancreatic secretion present; but the point that I want to make is that the absence of pancreatic secretion is a great help in diagnosis.

In regard to the technique that is essential, I have had the same experience as Dr. Crohn had in working with dogs at Berlin. He was struck by the action of the salts on the diastase. I did some work with great care, and noticed that the results were all wrong. I was using tap water, and found that there had been a contamination of the Berlin tap water and that they were using something to correct it. This made a difference in the results. I trained a man at Johns Hopkins to do this work. Every step counts in making this test. The same things must be done on each occasion. All of these things are absolutely fundamental. You must handle them with the most delicate care.

As for palpation of the pancreas, I have seen surgeon after surgeon fail and be unable to differentiate it. You can understand how difficult it will be to palpate through the abdominal wall and feel it.

I was interested in Dr. Libman's cases. The question of sprue is interesting. My feeling is that pancreatic achylia is the most im-

portant, and that the gastric achylia is merely incidental. My first case came to me from Ashhurst, of Porto Rico, and was an unquestionable case. It was in a young woman, whose home was in Virginia, and whose husband had gone from there to Porto Rico for work. She had gastric achylia, and I made a careful study of the case and reported it to this association. She did marvelously well on pancreatic therapy. Of course, we gave all the adjuvant factors; but the great thing in her case was the pancreatin. She now weighs one hundred and forty pounds. Once or twice a year, she comes up to have me look her over. Her gastric achylia has disappeared, but her pancreatic achylia is there. I can, in a week or two, bring back the symptoms of sprue by keeping her off pancreatin for that length of time; so my feeling is that pancreatic achylia is the significant thing, and that the gastric is only an expression of the toxin associated with sprue. Of course, regarding the question of the etiology, in mentioning duodenitis, gastritis and infections of the bile ducts, I did not go into the various preceding factors. I have not been convinced of the specificity of infection. I do not think that infections around the mouth do play a rôle in producing infection of the duct of Wirsung or the bile ducts.

One point that I do want to make is that if we are going to get anything out of ferment studies, it is necessary, in the first place, to establish the low normal. Anyone who has done work on dogs cannot fail to realize that there is a difference in the response of the pancreas to different food stimulants. I believe that it is quantitative—an absolute response to a definite meal. The difficulty is to obtain the total amount of the response and measure it. Constant variations below the low normal should make us suspicious; and absence within the limits of error should make us more suspicious of carcinoma or sprue. There may be cases in which pancreatic achylia is not present; but with a rigorous technique, which is essential, it is possible to reach a correct diagnosis. It is the instability of the ferment that makes the test so unsatisfactory in many hands. It should not be done by anyone except a skilled laboratory worker who absolutely checks up every bit of technique. I am a little more doubtful of the duodenal contents than of the stool, although it is much pleasanter to use the tube. Reduction or absence of ferments is of great significance in the early diagnosis of pancreatic disease.

THE NEUROLOGICAL BASIS OF THE COMMON GASTRO-INTESTINAL SYNDROMES FOUND IN PULMONARY TUBERCULOSIS.

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In discussing the relationship between inflammation of the lungs and altered function in the gastro-intestinal tract, it is necessary to recall the embryology of these structures; for this gives us a basis for understanding the manner in which they are related through the visceral nerves.

The respiratory system is formed from a diverticulum from the gastro-intestinal canal, and therefore carries with it the innervation of the mother structure, the same as the liver, pancreas and body of the bladder, which likewise have the same origin.

This embryological origin gives to the lung the same double innervation as that possessed by the intestinal tract. All smooth musculature and all secreting glands belonging to the lungs and bronchi are activated the same as the stomach and intestines (except the sphincters) by the vagus nerves which belong to the parasympathetic division of the vegetative system; likewise all are inhibited by the parasympathetics.

When the pulmonary structures are inflamed, sensory nerves belonging to both of these systems are irritated and result in reflex action. During the stage of toxemia the cells of the entire nervous system are irritated by the toxins. These express themselves, peripherally, largely through the sympathetic nerves and in this manner produce a general inhibition of action throughout the gastro-intestinal tract, relaxing the muscle of the wall. It may be that there is also an action of the toxins directly upon the muscles themselves, which interferes with their normal rhythm. The common symptoms on the part of the gastro-intestinal tract during toxemia are those of decreased motility and decreased secretion—hypomotility, hypochlorrhidria and constipation due to lessened secretion and lessened peristaltic action. This is common in all acute infections accompanied by marked toxemia.

The sensory sympathetics supplying the lung mediate with the

spinal nerves in the cervical segments of the cord and produce reflex spasm of the muscles supplied by the motor nerves; and reflex pain in the tissues supplied by the sensory nerves; and, if the process becomes chronic, trophic changes in the skin, subcutaneous tissue and muscles supplied by both motor and sensory nerves.

The sensory fibers of the pulmonary vagus (parasympathetics) mediate with other fibers of the vagus and with other parasympathetic nerves (VIIth and IXth cranial nerves) and with the Vth cranial nerve which stands in the same relationship to the sensory fibers of the vagus as the spinal nerves do to the sympathetics.

It is through the vagus (parasympathetics), then, that the reflexes take place which affect the gastro-intestinal canal when the lung and bronchi are inflamed. This parasympathetic stimulation is the cause of all the common reflex functional disturbances in the gastro-intestinal canal which result from pulmonary inflammation.

Before one can understand the reflex symptoms which arise from inflammation in any organ, he must first appreciate the fact that nerve cells in different individuals and in the same individual at different times show different degrees of irritability; and second, that a nerve cell will not discharge, and produce action until the stimulus is *adequate*. In other words, nerve cells are able to withstand a certain amount of stimulation without producing action, and this amount varies in different individuals and in the same individual under different circumstances. This accounts for the variability of symptoms as noted in a given disease, a fact which has always made diagnosis difficult. The diagnostician must remember when studying symptoms, that a given stimulus *has a tendency* to produce such and such an action but that this action may not occur because the stimulus may not be *adequate* to discharge the nerve cells upon which the action depends.

When the lung is inflamed, the sensory fibers of the pulmonary branches of the vagus are irritated, and stimuli are carried to the sensory nucleus of the vagus in the medulla whence they are transferred to other neurons with which they mediate, viz., vegetative fibers of the VIIth, IXth and Xth cranial nerves and somatic fibers of the Vth cranial nerve.

When mediation takes place with the Vth, VIIth and IXth cranial nerves reflex action results mainly in an increased secretion and an increased irritability of the nasal and oral cavities, the pharynx, salivary and lachrymeal glands; in vasomotor disturbance in the cheeks and tongue; in trophic change in the tongue which at times causes it, when protruded, to turn toward the affected side; and in pain expressed in the sensory neurons of the Vth cranial nerve (headache).

When mediation takes place in other portions of the vagus nerve, reflex action may result in any tissue which this nerve supplies, such as the larynx and pharynx, the bronchi, heart, upper portion of the gastro-intestinal canal, liver and gall ducts, and pancreas.

Reflex stimulation, *if adequate*, produces the action which normally belongs to vagus stimulation in these structures—an increased tonus in the muscles and an increased glandular secretion.

It is now evident that if stimuli which course centralward over the sensory neurons of the pulmonary vagus are transmitted to the efferent motor neurons of the vagus which supply the gastro-intestinal canal that they will *have a tendency*, reflexly, to cause an increased tonus in the musculature and an increased secretion in the glands. This is what we find clinically when the pulmonary tissue is inflamed in such chronic diseases as pulmonary tuberculosis. The “so-called” functional disturbances on the part of the gastro-intestinal canal which are so common in pulmonary tuberculosis are nearly all of this type.

Variability characterizes functional disturbances. These symptoms may be present at one time and not at another. Unless one is familiar with this characteristic he can not fully appreciate the gastro-intestinal symptoms in pulmonary tuberculosis.

The common syndromes on the part of the gastro-intestinal tract which are indicative of a preponderating vagus stimulation and which often result from reflex stimuli arising in other organs which are the seat of inflammation are nausea, vomiting, hyperchlorrhya, gastric hypermotility, colicky pains, spastic conditions in the intestines, notably spastic constipation, colitis, diarrhea and intestinal stasis.

This group of functional disturbances makes up a considerable proportion of the symptoms on the part of the gastro-intestinal

tract of which patients suffering from early active or chronic semiquiescent tuberculosis complain; and sends the patient to the gastro-enterologist as often, if not more often than to the specialist in diseases of the chest. In fact, the reflex symptoms which are caused by clinical tuberculosis before the advent of marked toxemia, and productive cough, and during the stage of quiescence, are practically all expressed reflexly through the vagus in systems other than the lower respiratory: in the larynx as irritation and cough; in the heart as an inhibiting effect producing instability; and in the gastro-intestinal canal in the form of the syndromes above mentioned.

These symptoms may be caused by conditions in which the vagus nerve cells are hyperirritable (vagotonia); by conditions which produce a marked stimulation of the vagus or a decreased stimulation of the sympathetics; by direct irritation on the nerve cells lying in the walls of the stomach and intestine; by reflex action in one part of the intestine, the stimulus coming from another part; or reflexly from inflammation in other organs. The most common source of reflexes coming from without the intestinal walls, in my experience, is the appendix, gall bladder, lung and eye.

It can be stated as a rule that functional disturbances on the part of the stomach and intestinal tract, are more commonly an expression of reflex action from some other organ than from a disease of the tube itself; and when the syndromes here mentioned present, the appendix, gall bladder and lungs should be carefully examined for the presence of disease; and eye strain should be considered. One must not forget, however, that so-called nervous individuals (vagotonics) are also prone to show this same picture of functional disturbance. The expression of "nervousness" in the gastro-intestinal canal is predominantly that of increased vagus stimulation. When vagotonics suffer from a pulmonary tuberculosis or a chronically inflamed appendix or a disease of the gall-bladder, then the symptoms on the part of the digestive system which result from vagus stimulation are prone to be very much exaggerated.

In order to understand what functional symptoms on the part of the digestive system are going to manifest themselves in a patient suffering from tuberculosis it is necessary first to know the nervous, the physical and the psychical condition of that

patient before he suffered from pulmonary tuberculosis, to know whether he previously suffered from any particular type of digestive disturbance, and to know what other complications may be present. It is also necessary to make a distinction between active pulmonary tuberculosis with marked toxemia and the type of the disease when toxemia is not prominent.

This is evident from the fact that reflex symptoms do not arise except as the stimulus causing the reflex is sufficient to overcome the action of opposing nerves. When nerve cells are in different degrees of irritability, the strength of stimulus necessary for their discharge will vary greatly. As long as the force of the sympathetic neurons in the intestinal tract is equal to or approaches the force of the neurons of the vagus nerve (parasympathetics) no disturbance in motility or secretion will take place and normal function will exist; but just as soon as the force in one system overbalances the other, symptoms will arise.

When the pulmonary parenchyma is inflamed as it is in tuberculosis, the sensory nerve endings of the pulmonary branches of the vagus nerve are irritated. This stimulus is carried to the medulla, where it is transmitted to motor neurons, which produce reflexes in structures supplied by them. Among the nerves involved are the motor fibers of the vagus which supply the stomach and intestine. This stimulus *has a tendency* to produce action in all the motor neurons to which it is transferred; and when it is *adequate* to overcome all opposing forces acting upon the sympathetics, it does so.

Whether a reflex gastric hypersecretion or hypermotility, or a reflex intestinal hypersecretion or hypermotility will occur depends upon whether the stimulus arising in the inflamed organ and transmitted to the musculature and secreting glands of the stomach and intestine through the vagus is sufficiently strong to overcome the opposing inhibitory action of the sympathetic nerves supplying those tissues. This will depend to a great extent also upon the natural characteristics and tendencies of the patient. That the stimulus is adequate in a large proportion of instances is evident from the frequency with which patients afflicted with pulmonary tuberculosis suffer from reflex nausea, vomiting, hyperchlorhydria, spastic constipation, colitis, intestinal stasis and colicky pains. These syndromes are common in patients who were free from them prior to their clinical tuberculosis and are

often rendered more annoying in those instances where they were previously present. I have seen hyperchlorhydria in many instances during the high fever accompanying cavity formation. Not infrequently have I seen a relative bradycardia at the same time. These symptoms can only be interpreted as being a result of reflex stimulation of the vagus from inflammation in the lung, coming as they do during severe toxemia, which stimulates the sympathetics depressing gastric secretion, and producing an accelerating influence upon the heart. Nausea and vomiting, which are often present at these times, are also frequently of reflex origin. These reflex relationships I have discussed quite fully in previous communications.^{1, 2.}

A few words in explanation of the reflex nature of these syndromes may not be out of place. I do not wish to be understood as maintaining that all *nausea and vomiting* found in pulmonary tuberculosis is of reflex origin; but I am sure that part of it is an expression of increased muscular tonus in the gastric walls. These patients also at times show more than ordinary degrees of hunger. Carlson has shown that this phenomenon is due to increased gastric motility.

Colicky pains are very common during the course of pulmonary tuberculosis. Unless there be a mechanical obstruction these are usually due to areas of spasticity in the intestine which interfere with the onward movement of the gas, causing it to accumulate, and distend the gut and cause the pain. When the intestinal musculature is in a state of increased tonus, different areas of the gut show different degrees of irritability, a condition which favors constriction at intervals and permits of the accumulation of gas with distention of the bowel proximal to the constriction.

Spastic constipation often results from reflexly increased tonus in the musculature of the colon, the stimulus causing the reflex emanating from the lung. This form of constipation is usually accompanied by colicky pains. It is due to the same cause, being an expression of the same increased muscle tonus (hypermotility).

Colitis in tuberculosis has the same reflex cause as the foregoing phenomena. The vagus when stimulated not only causes an increased muscle tonus leading to a hypermotility, but also an increased secretion in the intestinal glands.

The reflex type of *intestinal stasis* is caused by a retardation of

the intestinal contents in their progress through the canal as a result of spastic conditions in the bowel.

If we substitute inflammation in the appendix and the sensory fibers of the vagus which supply the appendix in the one case, and inflammation of the gall-bladder and the sensory fibers of the vagus which supply it in the other for the inflammation of the pulmonary tissue and the sensory fibers of the vagus which supply it; we have the mechanism which explains the gastro-intestinal symptoms in appendicitis and inflammation of the gall-bladder. Then, further, if we will substitute the stimuli which arise from eye strain, and the sensory fibers of the Vth cranial nerve which mediate with the motor neurons of the vagus, for the stimuli arising from inflamed pulmonary tissue and the sensory fibers of the pulmonary vagus we will have an explanation of the reflex mechanism through which eye strain produces symptoms on the part of the gastro-intestinal canal.

Through an understanding of visceral nerves and the relationships which are maintained by the various viscera through them, reflex functional disturbances are stripped of their former mystery and placed on an understandable basis.

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DISCUSSIONS.

DR. JOHN F. SAWYER, Cleveland: I was interested in the communication of Dr. Pottenger in regard to the relation between gastro-intestinal diseases and pulmonary tuberculosis. This subject has been of absorbing interest and importance to me for many years. We have almost a vicious circle established here, and while Dr. Pottenger has presented it from the point of view of the effect of the pulmonary condition on the gastro-intestinal tract, my attention was called to it from the other side. I think there is too commonly a failure in practice for us, as gastro-enterologists, to ascribe to these symptoms arising from pulmonary conditions their true importance; and I am sure that the treatment of tuberculosis has been rather step-motherly, as the Germans say, in the handling of the gastro-intestinal phenomena. The end product of the digestive activity is the manufacture of blood, which is highly important in the treatment of tuberculosis; and the power of recovery of the lung is dependent, in large measure, on the manufacture of suitable blood. I do not believe

that there is a diet for tuberculosis, but I believe that there is diet for tubercular patients, according to their particular requirements. In determining these, we have to take into account the psychic state. We have to remember the individual patient. The condition of the patient varies tremendously under the stress of apprehension, of family emotion, of financial conditions, etc.; and we have to treat these cases at their homes. The nerve phenomena of central, as well as of peripheral origin, due to reflex through those inter-communications of the nervous system, are matters which those who would treat the patient, and not the disease, must reckon with. I am sure that every such presentation as this, which sharpens the attention of physicians to the inter-relation of diseases, tends to bring these things more to the advantage of a great group of patients.

THE "GASTRO-INTESTINAL FORM" OF INFLUENZA.

BY JOHN A. LICHTY, M. D.,

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Notwithstanding the prodigious efforts which have been put forth in the past year during the recent epidemic of influenza to discover the specific micro-organism it must still be acknowledged that investigators are by no means in full accord as to the real cause of influenza. Any discussion, therefore, at present from a clinical standpoint may lead only to confusion. There is no doubt that the conception of certain "forms" of influenza which have been described in certain previous epidemics will necessarily have to be revised when once the etiology is definitely established.

About the close of the great epidemic of 1889-90 the Pfeiffer bacillus was looked upon as the specific cause of influenza. During that epidemic and even in epidemics previous to that time certain clinical manifestations have led to the designation of rather definite forms or varieties of influenza. Among these are the respiratory form, the gastro-intestinal form, the nervous form and other forms. For a description of these forms one may refer to the well-known article by Dr. O. Lichtenstern in Nothnagel's Encyclopedia of Practical Medicine: (American Edition of Nothnagel's Encyclopedia of Practical Medicine: Malaria, Influenza and Dengue; pages 523 to 716).

One would infer from this able clinician that in certain cases the respiratory system was the primary seat of the disease, in other cases the gastro-intestinal tract was the primary seat of invasion and presented definite pathology, while in still others the nervous system was primarily affected, to the exclusion of other organs and systems, and so on. This opinion has evidently been accepted by clinicians in the numerous, almost annual, sporadic epidemics which have occurred since 1889-90. This can be inferred from the abundant literature as well as from our recollections of descriptions, and of discussions of cases happening in our own times and experiences.

In one year it would be reported that the symptoms were largely of the gastro-intestinal tract, nausea and vomiting or diarrhea or even a most unusual incidence of acute appendiceal attacks; in

another the nervous system was the seat of the principal manifestations, extreme headache, depression, sleeplessness, etc. Because the Pfeiffer bacillus was found on the nasal mucous membrane, or even if it was not found, on account of an apparent epidemicity it was concluded the symptoms were indicative of a certain localized pathology and to be a form or type of influenza. These diagnoses insofar as our experience is concerned and insofar as we can glean from the literature were rarely verified by autopsy or by such definite bacteriological findings as we are accustomed to exact in disease from other causes.

If one will read carefully the description of the pathology given in Lichtenstern's article on influenza he will be impressed with the fact that it is chiefly and primarily of the respiratory system and not of the nervous system, gastro-intestinal tract, etc., notwithstanding his classification of "forms."

The pathology which he does describe as occasionally found in the gastro-intestinal tract such as a hemorrhagic condition, swelling of Peyer's patches, etc., could readily be explained as being coincident or secondary, as may be found in the course of any acute infection. It seems quite obvious, therefore, that the seat of the primary infection of influenza is in the respiratory system and tract. It has certainly been so in this past epidemic, as will be shown later.

This does not necessarily imply that epidemics in past years may not have differed in their symptomatology. All of us who have had experience with so-called influenzal epidemics will recall that in one year the symptoms were largely those of the nasal cavity and the associated sinuses, in another year of the tonsils and pharynx, in another year of the middle ear and eustachian tubes, in another year of the larynx and trachea, and again in another year as in the recent epidemic principally of the bronchial and lung parenchyma. But it was always of the respiratory system. In some epidemics it was so mild as to be almost imperceptible and apparently negligible, in others it was extremely grave. If it should seem desirable to classify the forms of influenza it would appear that such a classification, *i. e.*, depending upon the prevalent location in the respiratory tract would be more to the point and more desirable than classifications previously suggested.

All this is preliminary to the opinion that influenza is a disease

of the respiratory system and that manifestations elsewhere, at the onset, during or following the infection are evidences of the extreme severity of the attack, of definite complications, of sequellae or of intercurrent disease and not the evidence of the specific infection or pathology elsewhere.

In the recent epidemic every effort has been made by the essayist to find a case which might present the gastro-intestinal form of influenza. Among from five to six hundred cases in hospital practice under rather close observation and among a number of cases seen outside of the hospital quite a few had gastro-intestinal manifestations which might have led one to think at first that they were the chief and only symptoms, and that the infection and whatever pathology might be present was of the gastro-intestinal tract, but in none have we been able to show this to be the fact. In all the respiratory lesion was sooner or later recognized. Reference will be made to only two cases which are typical of others which were equally instructive.

One was the case of a lad of about 20 who had intense nausea and vomiting from the first with no other symptom but frontal headache and a conjunctivitis. He had a rather high temperature and the characteristically slow pulse. A diagnosis of influenza was made largely because of the prevalence of the epidemic. It was supposed to be of the gastro-intestinal form. An examination made by a competent nose and throat specialist revealed an ethmoiditis, with an accompanying conjunctivitis complicating influenza. The relief of the pressure in the ethmoid promptly relieved the nausea and vomiting.

Another was the case of a man who was sent to the surgical department of the hospital with a high temperature and a rapid pulse, suffering from pain in the left side of the abdomen and with an obstinate hiccough. The left rectus was rigid, peristalsis diminished, and there was definite tenderness on deep percussion over the left lumbar muscle. A perforation with peritonitis, or some lesion in the left kidney or ureter were suspected. The patient was referred to the medical department for diagnosis. In a few days definite evidence of pleurisy was found at the left base, later the complex and confusing signs of influenzal pneumonia were plainly present and the diagnosis of influenza established. The hiccough and rigidity of the abdominal muscles continued. It was supposed that there was a lesion below the dia-

phragm due to the influenzal infection. At the autopsy a purulent diaphragmatic pleurisy was found beneath the left lung. The lungs showed pathology characteristic of an influenzal pneumonia. The peritoneal surface and the abdominal organs were absolutely normal. Without an autopsy this case could easily have been passed, especially before the pleurisy was recognized, as one of influenza of the gastro-intestinal form.

Among our patients we had a number who had symptoms referable to the stomach which were characteristic of ulcer, or symptoms referable to the appendix but in nearly all we could obtain a history of previous digestive disturbances. In these cases we concluded that an old lesion had been activated by the influenzal invasion. This was, of course, not peculiar to the gastro-intestinal tract in influenza. Pathology in other organs and systems behaved in an apparently similar way. A striking example which must have been noted by all is that of the enlarged thyroids and the potential hyperthyroid patients. During or after the attack of influenza the thyroid became acutely enlarged and in many cases symptoms of hyperthyroidism appeared. Such an instance would certainly not warrant the designation of a "thyroid form" of influenza. No doubt many such cases have been thoughtlessly observed and the patients looked upon as manifesting the "nervous form" of influenza.

The autopsies done at the Mercy Hospital, where most of the clinical observations upon which this paper is based were made, did not reveal any characteristic pathology of the gastro-intestinal tract which could be attributed to the influenzal invasion, unless one considers the slight hyperplasia of the solitary follicles, which was noted by Dr. A. J. Brecken, the Assistant Pathologist. Dr. Klotz, director of the pathological department of the School of Medicine, University of Pittsburgh, who has done a goodly number of most thorough autopsies in connection with the work at the Magee Hospital, where the soldiers from the barracks were sent, reports no distinctive gastro-intestinal lesions. On the other hand, he reports besides the lesions in the respiratory system such as multiple abscesses of the kidney, characteristic lesions of bones and joints and of the long muscles of the body. Dr. H. H. Permar, Major R. C. Base Hospital No. 27, from the University of Pittsburgh, has reported to the essayist personally that in 120 consecutive autopsies in influenza cases, no specific pathology was found in the gastro-intestinal tract.

From the clinical and pathological standpoint accordingly we feel there is not sufficient data to warrant the designation of a gastro-intestinal form of influenza. We are aware that many cases are being reported as of this type and the question may be asked what is the explanation to this abundant evidence? It must not be forgotten that the incidence of influenza in some communities was as high as 40 per cent., and also that a lowering of physical reserve would naturally favor the progress of inter-current disease in any organ. It should not be difficult, therefore, to find a half dozen cases of appendicitis or so many more cases of acute exacerbation of a latent peptic ulcer in any community among the 40 per cent. stricken.

It is the opinion of the essayist, therefore, that the symptoms of the so-called gastro-intestinal form of influenza can all be explained by the well-known intoxications which may be associated with an acute infection, or by previously existing pathology in the gastro-intestinal tract, or by the occasional reference of symptoms to organs below the diaphragm from pathology immediately above the diaphragm or by the mere incidence of the usual diseases of the gastro-intestinal tract in such a widespread epidemic.

The gastro-intestinal manifestations of influenza in this epidemic and possibly in past epidemics whether they occurred during the attack or afterwards are complications or sequellae and are not evidence of a distinct form of influenza.

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DISCUSSIONS.

DR. SIMON, New Orleans: All of us came out of the epidemic of influenza last winter with some very definite ideas in regard to the disease. As one of the generation who had no occasion to observe the clinical side of previous epidemics, my mind was open, and like Dr. Lichty, from the very beginning I had my observation keyed up for the gastro-intestinal aspects of the disease. I looked in vain for the group of cases which had previously been designated as belonging strictly to the gastro-intestinal type. While digestive symptoms prevailed in some cases, they did not take marked precedence over the usual clinical features of the disease. Influenza undoubtedly produces a neuro-muscular toxin, which affects the organs of digestion in common with other organs. For that reason it is not at all surprising, it is rather to be expected that one should find both in the midst of the acute manifestations of influenza and in the period that follows a tendency to disturbances of gastric motility and sensation. Gastric

atony, in fact, I have found, has constituted the most common aftermath of the acute attack and one that has persisted in some instances for many months.

Another of the sequellae I observed in two instances was what I was forced to call, for want of a better name, Influenzal Gastralgia. In two cases I had occasion to see a patient suddenly seized with severe pain in the epigastrium, coming on in both instances two or three weeks after recovery from the acute attack. One of the patients had three such attacks with intervals of several days between, and each time I was compelled to administer an opiate. With each attack there was evidence of shock along with a very rigid abdomen suggesting at first the presence of an acute surgical abdomen. The condition cleared up, however, so readily with the single injection of morphine that all thought of surgical complications was soon dispelled. I am not a great believer in idiopathic gastralgia, as the term is ordinarily applied; but in this case I think it necessary to have recourse to some such designation as one of the possible complications of influenza.

DR. PILCHER, New York: I was in Paris at the time the epidemic hit us abroad and the men were being sent back in a pretty bad condition. We had two wards set side by side, each of them containing 100 beds. I and my officers at this hospital had charge of one ward and another set of officers the adjoining ward of a hundred beds. The men were entered as they came alternately one to one ward, and one to the next; so that each ward had practically the same class of cases. In the adjoining ward of 100 cases, 80 of the men died. In the ward under our jurisdiction there were no deaths. In looking up the treatment that the physicians were giving in the other ward I found it was the usual treatment of a little acetanilid, a little sodium salicylate, caffein and camphor monobromate or simply asperin alone.

Working on the principle that it was a streptococcus infection and that salicylic acid had given good results in combating streptococcus infections in other conditions, such as rheumatism, we decided to give these patients as an initial dosage five grains of calomel and 80 grains of sodium salicylate, which was repeated at the end of six hours if the temperature still remained high. None of them ever received more than two doses, and as a rule they were perfectly convalescent at the end of 48 hours and none of them showed any signs of salicylism. I am wondering if any of the men over here in the states have used such massive doses of sodium salicylate, in treating this condition, and what their experience in its use was.

DR. SAWYER, Cleveland: I want to say with reference to Dr. Lichty's most interesting communication that the cases which I saw, which did most satisfactorily, were cases which received relatively large doses of sodium salicylate, though not as large as that indicated. Customarily I give 20 grains every two or three hours, and was fortunate in seeing, I think, fewer of the severer cases with developments. How

much to associate with this I don't know. I do not feel that I am convinced that there can be any gastro-intestinal phase of importance to ascribe to the influenzal influence, because there is no pathological change in the tissue. Believing as I do that the metabolism of the body and the chemical changes which go on are to such a profound degree influenced by the nervous system, I feel as Dr. Simon says that the toxin may produce functional phenomena which may be dominant in the clinical course of the case. That there are lesions to be recognized post mortem, I am in no position to either assent or withhold assent. I could not express an opinion. But I feel very sure that, as has been indicated in the discussion, a toxic process affects profoundly nerve centers which control enormously important functions of the body and may take precedence over the symptoms which perhaps are directly to be attributed to a focal or cellular change.

DR. PILCHER, New York: We unfortunately did not have time or opportunity to work out the above mentioned finer points. There was very little of the gastro-intestinal type, such as has been described by Dr. Luria, in Paris. These men all came back from the trenches seriously sick. They were all from the Chateau Thierry sector, where they had been stricken. Practically all had been 48 hours without attention before we saw them. They had their broncho-pneumonias already started, and those 80 men that died in the pavilion adjoining us developed a typical haemolytic broncho-pneumonia, to which they succumbed. They were not in a condition where you could afford to temporize by giving small doses. The treatment had to be heroic or practically nothing. What the subsequent course of these men who did convalesce was I cannot say, because they were evacuated as quickly as they could be moved without danger to the back areas.

DR. BASTEDO, New York: In two of my cases at the City Hospital, which had jaundice, too profoundly ill to do anything definite to the alimentary tract, the autopsy showed a follicular duodenitis without any special involvement about the papilla of Vater, but the whole second portion and more or less of the third portion of the duodenum was granular. I am almost sure that out of the nearly 50 autopsies which we had at the City Hospital there were two other cases that showed a somewhat similar picture, and this interested me greatly because it is obvious that the jaundice might have resulted from a very definite inflammation localized in or about the bile passages.

DR. LICHTY (closing): There were two reasons for my suggesting this paper, and the primary reason is to fulfill my obligation to the new secretary, and my second reason was that in a rather wide consulting practice I found a number of instances where the nausea was interpreted as being primarily due to gastro-intestinal disturbances, and at the same time neglecting the pneumonia or influenza which they might possibly have handled more satisfactorily with

more direct and specific treatment. With reference to salicylate, I might say that in our experience we used the salicylates and bicarbonate of soda in rather large doses; but we did not have such striking results as Dr. Pilcher has had. Now with reference to the Brooklyn epidemic: during this epidemic patients became so suddenly ill and died so quickly that I do not think there was time enough for accurately diagnosing intestinal lesions as differentiated from the general infection.

I might also add that I spoke with Captain Nicholson, who had charge of the pathological department of one of the camps out West—in Kansas—and in 125 consecutive autopsies which he made he told me that he found nothing at all in the gastro-intestinal tract as a primary lesion, characteristic of influenza.

Might it not be possible that in some of these cases where the gastro-intestinal symptoms were so definite that this center, which was described this morning by Dr. Alvarez may have been intoxicated, as Dr. Simon has described, and they might have had gastro-intestinal disturbance, nausea and vomiting from the disturbance of the nervous mechanism through the toxin?

FOOD CONDITIONS AND NUTRITIONAL DISEASES IN
EUROPE.

BY LIEUTENANT-COLONEL SEALE HARRIS,

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Since I shall discuss the work on gastro-enterology in the United States Army during the World War before one of the sessions of the Section on Gastro-Enterology and Proctology during the meeting of the American Medical Association, and since practically all of the members of the American Gastro-Enterological Association will be present at that time, I shall not take up your time now to dwell upon the accomplishments and the sacrifices of gastro-enterologists to serve their country in the greatest crisis of our history. I would like to say, however, that the patriotism manifested by members of the American Gastro-Enterological Association has not been exceeded by the members of any other specialty in medicine or surgery. Though many of our members are over age, and others belong to the teaching staff of medical colleges, whom the Surgeon General declined to commission in the Army because the training of medical students was considered essential war work, the members of this Association never failed when called upon to make any sacrifice for war work.

I would like to mention particularly the work of Dr. Reh fuss, who gave up much of his time, at considerable expense, for several months, in his efforts to advance the specialty of gastro-enterology. Dr. Gerry Morgan, though he does not like to admit that he is over the military age, helped in the work in every way possible. I needed a great deal of advice and assistance while at the head of the Section on Gastro-Enterology in the Division of Internal Medicine, and since Dr. Morgan lived in Washington, I had to call upon him frequently and he always responded promptly and heartily.

I feel that I should also say something regarding the effort of the man whom we regard as the Dean of gastro-enterology in the United States, Dr. Max Einhorn. Dr. Einhorn is unwilling to admit that he is over thirty, and when the call came, he gave up his work and accepted a commission as captain. I would like

to take this occasion to say that I protested at this commission being given a man of Dr. Einhorn's eminence, but what I and other Medical Reserve Corps Officers had to say regarding the question of rank was overruled. Dr. Einhorn did not mention rank, and went into the service at the first call. He was assigned to duty at Camp Upton and very soon afterwards received an injury to his knee, which disqualified him for service. He continued to serve, however, longer than he should have before giving up and he was disabled for months on account of it.

I would also like to mention the service that was rendered by quite a number of other men in gastro-enterology, but time will not permit my going into a discussion of the many splendid things that were done by individual members of the American Gastro-Enterological Association. It is enough to say that all of you did your duty, as you saw it, and too much credit or praise cannot be accorded to the men of our specialty for their part in war work.

During ten months of service in Europe, I had the good fortune to visit France, Belgium, England, Italy, Austro-Hungary (Trent, Triest, Istria, Fiume, etc.) and Germany. The relation of food conditions to nutritional diseases in those countries interested me tremendously, and it occurred to me that other gastro-enterologists would like to hear something of the results of my investigations on this subject.

FOOD AND FIGHTING EFFICIENCY.

In discussing "Food Conditions and Nutritional Diseases in Europe" it may be well to mention, first, that the American Armies, both in the United States and in Europe, throughout our participation in the World War, have been the best fed troops in the history of warfare. It is true that our army rations have contained an excess of proteins (meats, beans, etc.), but otherwise the soldier's diet has been well balanced, with plenty of good wholesome food for everyone.

The selection, purchase and transportation of food for an army of more than 2,000,000 in a foreign country, 3,000 miles from home, and the preparation of this food under conditions in which our troops fought in France, is surely among the great achievements of the war. Napoleon is credited with saying that "an army moves on its stomach"; and the fact that the American

troops were so well fed may account partly for the efficiency and the eagerness with which they fought, always moving at a rapid pace, towards Germany.

I had the privilege several times of eating with our soldiers at the "Front," and in various other places in France; and they were always well supplied with food, except on the occasions when sometimes in the midst of battles food had to be carried to them at night. I also had the opportunity of observing the meals that were served the French and British soldiers, on several occasions. They were not so well supplied with food, nor was it so well prepared as for our men.

It is not entirely irrelevant to say that the American troops were the finest looking soldiers in Europe, because the reason that they were such perfect specimens of physical health was partly due to the fact that they were always supplied with good wholesome food. The greatest thrill that I have ever had—and one that I felt many times—was to see our soldiers marching in France and Germany. I saw them marching to the Front, returning to rest camp after battles, and in various parades; and it was always with the same thrill and pride that I watched them.

The fact that the Allies had much more food than the German and Austrian troops was a great advantage to us. On one occasion, last October, in Rheims, I saw 4,000 German soldiers the day after they were captured by the Americans and French in the fighting around Grand Pre. They showed unmistakable evidences of being undernourished. Later, in Germany, I was told by a medical officer in the German Army that it was a fact that for the last few months of the war, even though the German soldiers were better fed than the civil population, they did not have food to keep them in fighting condition.

Corroborating the statement that the United States Army has been well nourished during the World War is a recent report of the War Department which shows that American soldiers gained, on an average, 12 pounds during their recent service in the Army. Contrast this with the fact that in the Spanish-American War our soldiers lost on an average of 22 pounds each, and the difference is startling. It should be said, however, that the average loss of weight in the Spanish-American War was not all due to lack of the proper food; because nearly 20 per cent. of American soldiers in that war had typhoid fever and

were discharged from service before they had regained their weight. The difference of 32 pounds in the average weight of American soldiers after serving in the Spanish-American War and in the World War, is a great tribute to the improvement in the efficiency of the quartermaster's and medical departments of the United States Army.

FOOD INFECTION AFTER GERMAN RETREATS.

The care with which our food was protected from infection also speaks well for American efficiency. During my service in the Surgeon General's Office when in charge of the Section on Gastro-Enterology in the Division of Internal Medicine, I inspected the work in digestive diseases in a number of base hospitals, and had reports from the gastro-enterologists in all of them—and not one case of food poisoning, or food infection, was reported. I did not learn of any cases of food infection in France, except during the "drives" when sanitary conditions were found to be particularly bad in the territory that had been occupied by the Germans. After the German defeat and retreat in the Chateau Thierry region they are said to have left things in a frightful condition.

Colonel Grissinger, in discussing "Feces Disposal and Fly Prevention in a Division,"¹ at the September meeting of the Research Society in Paris, gave a graphic account of the horrible stench left by the Germans in their retreat. He said that they left horses and men dead and putrefying on the battlefields in the hot sun for days; and they seemingly tried to infect everything that they could not take with them. They polluted the wells and streams; and defecated on the floors, tables, and chairs, and even in bureau drawers; and everywhere they thought they could ruin furniture and houses, so that nothing could be used by the victorious troops of the Allies. Our soldiers said that it was a great deal easier to "clean up" the Germans in fighting than it was to clean up after them when they had been whipped. As a result of the filthy habits of the Germans, flies were swarming everywhere, and it was impossible to keep them away from food.

From various enemy reports which were secured by the Allies, and the visible manifestations of dysentery, which the Germans left behind them in their retreats, their soldiers must have suffered more from dysentery than did those of the Allied armies. It is surely a fact that until the end of summer wherever our armies

chased the Germans, in spite of every precaution that rapidly advancing troops could make, they developed epidemics of dysentery. These epidemics were of the bacillary type, the Flexner-Shiga bacilli being the principal offenders, though often there were mixed infections.

Zinsser,² at the September meeting of the Research Society, reported that among the thousands of cases of dysentery among American soldiers, the paratyphoid bacilli had been found in a few cases. He had also observed in the feces of a number of cases a new bacillus closely resembling the typhoid group. Siler³ at the same meeting said that in a number of cases of dysentery among American soldiers in France the *entameba histolytica* had been found. There were thousands of cases of nausea, vomiting and diarrhea, usually ascribed to food infection, or to contaminated water, after the Chateau Thierry drive. They were usually mild and subsided after a few days' rest with restricted diet. There were, however, many cases of severe dysentery.

FOOD CONDITIONS IN FRANCE IN 1918-1919.

Food conditions among the civil population of France in 1918 were serious, though not so bad as I expected to find them. When I arrived there in May it was not possible, at least in hotels, to get milk, butter or other fats and sugar. Bread, which was heavy and unpalatable, could be obtained, but not in sufficient quantity. Apparently there was plenty of meat, but all the fat was cut off before it was sold, and it was very expensive, so that the poor could not buy it. The delicious French rolls, cakes, pastries and other gastronomic delights that are happy memories to those who had been to France before the war, were not to be had at any price. France was surely on war rations in 1918.

My personal experience illustrates the food situation in France last year. After having lived for three months in what is considered one of the best pensions (boarding houses) in Paris, I weighed and found that I had lost ten pounds. I mentioned this fact at the table, which seated twenty-five or more, where there were a number of American, French and Italian officers, and found that all of those, who had been in France for some time, had lost flesh in amounts ranging from ten to forty pounds. We were getting the nicest anti-fat diet imaginable, but it was typical of what was served everywhere in France. All our food was

nically prepared and none of us realized that we were not getting enough to eat until we found that we were losing flesh.

The French breakfast ordinarily consists of rolls, butter and coffee or cocoa; but we had only two thin slices of war bread, no butter, no milk or cream for our coffee or cocoa, and it was sweetened with saccharin. We were, therefore, consuming less than 300 food calories for breakfast.

For luncheon we had plenty of vegetables, potatoes, rice, meats (particularly beef, veal and fish) and fruit, oranges, figs or grapes. Dinner was the same as luncheon except the addition of a thin soup. We had no milk, butter, sugar or desserts, except fruits—usually grapes. I estimated that we were getting about 2,000 calories of food a day, one-fourth of which was proteins—not a well-balanced diet, or one on which health and efficiency could be maintained for many months. I, therefore, moved to the Officers' Club (Hotel du Palais) in charge of the American Y. M. C. A., which provided us with everything that the French markets afforded, and, in addition, butter, sugar, canned milk and plenty of bread. I soon regained the flesh that I had lost and felt more like performing the work that I had to do.

NUTRITIONAL DISEASES IN FRANCE.

There can be no question but that for two or three years the whole French population has been undernourished, particularly the poor, and as a result the general death rate, especially among children, has increased considerably. Tuberculosis has increased enormously among the civil population of France. The same is true of the anemias. Scurvy, particularly the infantile variety, which was said to be rare in France prior to the war, is now a frequent condition. Epidemics of Vincent's angina, dysentery, and the infections of childhood, are said to have been worse than ever known in France. I could find no one in Northern France who had seen pellagra, but I did not visit the Southern districts, where it formerly prevailed. It should be mentioned that the American Red Cross has saved thousands of lives and has prevented many more thousands of cases of illness by distributing food and in establishing free clinics all over France.

The shortage of fats, sugar and wheat products in France lasted up to several months after the signing of the armistice. Conditions were improving when I left France in February, but

food was still so expensive that the French complained that they could not buy it. Incidentally, the French blame the Americans for raising the price of food and everything else that is sold in France.

FOOD CONDITIONS IN ENGLAND.

I was in England in June of last year, and apparently there was a more serious food shortage there than in France; and there is no doubt but that the British suffered greatly because of their inability to get sufficient fats, meats and sugar. While there was apparently plenty of meat everywhere in France, it could not be obtained, except on cards and then only in small quantities, in the hotels and cafes in London. Fish, however, was available. I was in England for only a week and my work was such that I had but little opportunity to investigate food conditions and nutritional diseases, but am convinced that the British suffered more than the French from the shortage of food.

THE ITALIAN WAR DIET.

Food conditions in Italy were worse than in either France or England. I spent three weeks there in January of this year, and from the information that was obtained from Red Cross workers, and from many Italians, I am convinced that there was a very serious shortage of meat and wheat in Italy in 1917 and 1918. Indeed, I was informed that the disaffection in the Italian Army, which resulted in the disastrous retreat in 1917, was largely due to the fact that the Italian soldiers, and the civil population of Italy to a greater extent, were not getting enough food. I was also informed that the American Red Cross had much to do with keeping Italy in the war, by furnishing her people with food during the great crisis through which that country passed in 1917.

The meat shortage in Italy was said to have been most serious. Even two months after the signing of the armistice meat could not be had at many of the hotels and it was so expensive that the poor could get but little. The fact that the Italian Government prohibited the manufacture of macaroni and spaghetti is enough proof of the scarcity of wheat. The only macaroni I saw in Italy was in the American Hospital at Vicenza. This hospital, which received wheat from America, had a macaroni ma-

chine which was said to have been worked overtime; and partly for this reason the sick and wounded Italian soldiers—the “macaronis” as the American “doughboys” called them—wanted to be treated by the American surgeons in Italy.

THE INCREASE IN SUICIDES AMONG GERMANS.

The suicide rate in Germany has always been high, but since the armistice it is said to have increased enormously, particularly among the professional classes. The cause of this increase in suicide rate is thought to be the lack of nourishment and the despair of thinking men and women in Germany, who see no future for themselves or for their country.

Quite a number of prominent physicians in various lines of work are said to have committed suicide. Among them was Dr. Adolph Schmidt, whom most of you knew, and whom I think that all of us have considered as one of the greatest authorities in the world on intestinal diseases. I regarded him as one of the most courteous gentlemen and one of the ablest physicians whom I have ever known. I shall never cease to appreciate his kindness to me. While working in Berlin, in 1906, I had been informed of Dr. Schmidt's wonderful work in intestinal test diets. En route from Berlin to Vienna, I spent several days in Dresden and Dr. Schmidt, without fee or hope of reward, gave up much of his time to show me the work that he was doing. It was, therefore, with genuine sorrow that I heard of the tragic end of this great man, even though he was a Colonel in the Medical Department of the German Army. I have wondered, too, what has become of many others of the great physicians in Germany and Austria, at whose feet we sat in our studies abroad. No doubt many of them are living but alas! we can never again go to them for the knowledge and inspiration that has been responsible to a very great extent for developing the science and practice of gastro-enterology.

COMPENSATIONS TO THE GERMANS FROM A LOW DIET.

Even starvation has its compensations and German physicians say that since over-eating has not been possible since 1916, there has been a decrease in stomach diseases, but that when a gastric disorder does arise, it is very difficult to relieve, because the proper diet is lacking. I was also informed that the low diet

has helped a great many people who were accustomed to over-eating before the war; and that liver diseases, chronic nephritis (Bright's disease), and other chronic ailments which affect men and women over 50 years of age, have decreased considerably. Diabetes, a disease which in many cases is thought to be due to over-eating, and in which the best treatment is starvation, is said to have disappeared from Germany. I asked a physician about gout, a disease formerly much dreaded by the Germans. He laughed and replied, "We don't have gout any more."

I heard considerable complaint about the beer which the Germans are getting. They said that there is no "substance" in it. It was described as "colored, brown water, a mysterious stuff that people can drink any amount of and never get crazy." One man, however, said that the German people were enormously better off because they cannot get their beer.

The Germans have had no coffee for three years. They have prepared substitutes of parched acorns, or burnt barley and chicory, which are not unlike genuine coffee in taste and appearance, but lack the "kick" of coffee because they are free from the stimulant, caffeine, which is the active principle of the coffee bean. Caffeine is really a powerful drug, and, therefore, excessive coffee-drinking, such as was the German habit before the war, is harmful. Since coffee has practically no food value, except from the sugar and cream with which it is usually taken, the Germans are fortunate to be freed from the caffeine habit. They have tea which also contains caffeine, but few Germans seem to have the tea habit.

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ACUTE PANCREATITIS (PANCREATIC NECROSIS) BASED UPON PERSONALLY OBSERVED CASES.

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This type of pancreatic lesion is less frequently observed than that of chronic pancreatitis and carcinoma.

Although the literature is rich in excellent contributions, the majority of physicians and surgeons frequently neglect to think of acute pancreatitis when confronted with a patient suffering with an acute abdominal lesion, and when the abdomen is opened the surgeon often fails to look for the most characteristic evidence, fat necrosis. And even when he finds this evidence, he is somewhat in a dilemma as to what constitutes the best method of treatment. Two recent cases observed in February and May of 1919 have stimulated me to review again all of my records of cases of this lesion and carefully to go over the notes on my own cases and those in the literature which were very complete up to 1904.

CASE REPORTS.

CASE I.—Pathol. No. 22012. Acute pancreatitis. Symptoms of ten hours' duration. Operation: drainage of the lesser peritoneal cavity through a mid-line incision and drainage of the gall-bladder. Recovery.

This patient was referred to me by Dr. Talbott, of Baltimore. She was a white female, aged fifty-two, fat and overweight, but her general health was excellent. Some twelve years previously her gall-bladder had been drained and an impacted stone removed from the cystic duct. For more than eleven years she had been free from any attacks of indigestion and of gall-bladder colic from which she had suffered for a number of years before that operation.

During the last three months before the present attack, she had had a return of her old indigestion, but nothing like acute gall-bladder colic.

In these recent attacks of so-called "indigestion" the pain was in the epigastrium on both the right and left side, associated with some belching of gas. Although she consulted a physician, the attacks were so slight that nothing was done beyond the administration of some simple medication.

Present Acute Attack.—Ten hours before admission to St. Agnes Hospital the patient was awakened out of a sound sleep by an intense pain in the epigastrium, present on both sides and somewhat of a girdle character. She immediately vomited the undigested evening meal, which had been eaten without discomfort and with relish some six hours previously. Since then, she had vomited three times a clear, bile-stained material. In those ten hours she had received one and one-half grains of morphine—one-half grain by mouth and the same dose twice hypodermatically. There had been no movement of the bowels. On admission to the Hospital the patient was suffering pain, but less acutely in character. She was positive that it was on both sides of the epigastrium and often went around the entire body below the diaphragm, of a so-called girdle character. At this time, and later during her convalescence, the patient informed me that in all her gall-bladder attacks previous to the operation twelve years previously she felt certain that she had never had an attack similar to this nor suffered so intensely from pain. She also without suggestion volunteered the information that she had never been so prostrate in her old attacks as she was in this one.

Examination.—The patient was not cyanotic, a point emphasized by Halsted. She looked, however, ashy, which might have been due to the large dose of morphine. The pulse was 88, the respirations about 26, chiefly thoracic; the blood pressure 120-80. There was no jaundice. The patient stated that she felt prostrated and nauseated. But she lay quietly on her back with the lower limbs extended. The abdomen showed no marked distention except from fat. The upper halves of the recti muscles were held rigidly and over the epigastrium there was some tenderness. There was no increased sensitiveness in the region of the gall-bladder. The lower recti and lateral abdominal muscles were fairly well relaxed. The area of liver dullness was present and there was no dullness in the flanks.

The temperature per rectum was 100°; leucocytes, 13,600; polymorphonuclears, 71 per cent.; r. b. c., 4,350,000; hemoglobin, 90 per cent. The specific gravity of the urine was 1.030; it was acid and gave no reaction for albumin and sugar. Gastric lavage removed a large amount of brown-stained fluid which showed a total acidity of 3; no HCl; bile was present.

We had evidence, therefore, that the contents of the duodenum with bile were regurgitating slightly into the stomach, although it was sixteen hours since any food had been taken, and ten hours since the vomiting began. An enema brought away a small amount of bile-stained fecal matter.

Clinical Diagnosis.—I felt quite positive that we were dealing with a pancreatic lesion.

Operative Pathology.—The operation was performed two hours after admission, about twelve hours after the onset of the first pain. The patient took the anesthetic, ether, quietly, and during the operation the pulse rose from 80 to 120 and the blood pressure varied from 120 to 130. The full anesthetic time was one hour and twenty minutes.

Exposure of Gall-Bladder.—I decided that it was wiser to expose the gall-bladder through a right rectus incision for drainage and also to ascertain whether any stones were present. In the thick subcutaneous fat there was no fat necrosis. On opening the peritoneal cavity adherent omentum was encountered. This was apparently huge. In it I could see no fat necrosis and no signs of hemorrhage, but the omentum was infiltrated with serum, having the appearance as if recently injected with salt solution. We could observe in the upper portion of the wound the blue, distended gall-bladder surrounded by this adherent and edematous omentum. The liver above appeared normal in size and color. These omental adhesions were not disturbed. Although it was twelve years since the drainage of this gall-bladder, it was still adherent to the parietal peritoneum.

(It is important to note here that in spite of these adhesions the patient had remained free from any discomfort for a period of more than eleven years.)

When the gall-bladder was opened, the wall was thin and the contents, bile, appeared normal. No stones could be found. Above and to the left of the gall-bladder there was an opening through which I could see the stomach, and this organ was not distended, and when I palpated this area it was not adherent. My finger could pass over the pylorus down over the duodenum to the right. There were no adhesions there.

I felt confident that there was, from the clinical picture, a pancreatitis with fat necrosis. The edema and infiltration of the omentum was to me a new phenomenon, but this was the earliest case that I had had the opportunity to observe or read about.

A tube was sutured into the gall-bladder with chromicized catgut and the right rectus wound was closed with buried catgut and silver wire through-and-through.

Exposure of the Pancreas.—An incision was made in the mid-line above the umbilicus. The subcutaneous fat was not so thick and there was no fat necrosis. On opening the peritoneal cavity there escaped a little clear fluid, not the brown-stained peritoneal exudate so characteristic of the later hours or more fulminating form of pancreatitis. The omentum exposed in this area was not adherent, and I could see neither fat necroses nor areas of hemorrhage. The stomach was seen. It was not distended. Bringing the greater curvature of the stomach into view, a point for perforating the omentum and gastro-colic ligament was selected 3 cm. below the stomach in a non-vascular area. After passing through fat at least 4 cm. thick, I exposed and nicked a membrane resembling peritoneum, and then there escaped a blood-stained fluid, but not the characteristic dark-brown fluid already mentioned. The lesser peritoneal cavity had, therefore, been opened. Gently enlarging this opening with retractors and sponging out the fluid, I could feel and see in the depth the pancreas. It was covered by the transparent posterior peritoneal membrane. Its definite markings could be recognized, and the pan-

creatic tissue, instead of being white, was of a livid pink, and on the peritoneal covering I could see and feel three sulphur-colored granules, so typical of the fat necrosis. The clinical diagnosis was therefore confirmed, and I had seen for the first time the earliest stage of the so-called acute diffuse pancreatitis. The area of pancreas seen was not much larger than a silver dollar, while the area palpated was very much larger. But I did not palpate either the head nor the tail of the pancreas, as this would only have produced more trauma. Nor did I pull out the omentum and transverse colon in order to inspect and palpate the pancreas through the mesocolon. The colon and small intestines were not seen during the operation. The edema of the omentum was much more marked in the area about the gall-bladder than in the area through which the pancreas was exposed.

I did not make incisions into the pancreas as recommended by von Mikulicz, and followed later in this country by Porter, because in my experience drainage of the lesser peritoneal cavity down through the pancreas had apparently been sufficient.

Drainage Through the Lesser Peritoneal Cavity.—Through the opening I passed a large rubber drainage tube to allow the escape of any fluid and surrounded this by strips of iodoformized gauze in order to isolate, wall off and make a direct fistula between the skin and the lesser peritoneal cavity down to the diseased pancreas. The wound above and below this drainage was closed with silver wire. In both subcutaneous fat wounds two small Carrel rubber tubes were placed in order to facilitate the escape of necrotic fat. If one does not provide for the escape of this material, the entire subcutaneous fat wound is apt to break down, and subsequent events in this case demonstrated the value of this protective drainage.

Post-Operative Convalescence.—Twenty-one days after the operation the patient showed every evidence of a complete recovery. When she came out of the anesthesia her first remark was, "You have relieved me of my pain." The tube in the gall-bladder had drained bile and came away spontaneously on the fifteenth day. There was still a slight discharge of bile from this wound. There was some discharge of necrotic fat from the lower angle of this wound.

The drainage from the lesser peritoneal cavity was of a serous character and slight. The larger tube and gauze had been removed on the twelfth day and two smaller Carrel tubes had been introduced. These tubes were irrigated daily with salt solution. Beginning on about the fourteenth day, the patient had an evening temperature of about 102° and some epigastric discomfort. Two days later when irrigating this wound, there was removed four or five ounces of a white granular material resembling broken-down fat, and then twenty-four hours later more of this material. Since that time there was no fever and no discomfort.*

*July 31, 1919. It is now about two and one-half months since the operation, and for about three weeks there has been little or

REMARKS ON CASE I.

Based upon my own experience and the literature, I have come to the conclusion that this type of drainage of the lesser peritoneal cavity is of the utmost importance. I have not as yet sufficient evidence that this alone is curative in the fulminating fatal type of the so-called acute hemorrhagic pancreatitis. But this form of drainage does relieve the accumulated fluid in the lesser peritoneal cavity and thus relieves tension, and it provides for the later escape of necrotic fat and of necrotic pancreatic tissue. In some of my cases so drained there has been no escape of any material except serum; in others material similar to that in this case, while in still other cases masses of necrotic tissue have come away which, when studied microscopically, showed the connective tissue framework of the pancreas.

In my own series this is the earliest case subjected to operation, and I have yet to find an earlier one in the literature. It differs from all the other early cases which I have observed or read about in the absence of any bloody or brown exudate in the general peritoneal cavity, and in the absence of fat necroses until the pancreas had been exposed in the lesser peritoneal cavity.

This may be explained either by the fact that in this case the pancreatitis was less acute, or it was exposed to view by an earlier operation. The recovery in this case is no proof that very early intervention will cure all cases of acute pancreatitis of this and the more fulminating type, but it does prove that it provides for proper drainage should the pancreatic necrosis go to the stage of suppuration with the formation of a pancreatic or peripancreatic abscess. My study of the problem seems to show that pancreatic abscess never recovers without operation, and

no discharge from the sinuses and there has been no discomfort. Between my last note on the twenty-first day and for about three weeks the patient had attacks of epigastric discomfort with fever which were relieved by dilating the sinus and evacuating purulent material. That is, it was almost two months before the evidence of necrosis ceased. This observation demonstrates the importance of keeping the external wound open until there is every evidence that suppuration and necrosis of the tissue in the depth of the wound have ceased. As we have no method of estimating the extent of the pancreatic necrosis, we are unable to estimate the duration of complete healing. For this reason the surgeon should personally supervise the care of the draining sinus. Apparently the external wound has a tendency to heal before the healing of the deeper wound is complete.

August 10, 1919. The patient is clinically well, but the sinus is not yet healed.

there has been a large mortality in late operations. This evidence should be sufficient to indicate our attitude toward acute pancreatitis when observed and recognized in its early hours.

The problem is: what shall we do in those cases of the fulminating type which rapidly develop the clinical picture of shock in the first twenty-four hours. One group of observers advises immediate operation irrespective of shock; others advise delay, fearing that the additional shock of the operation will lessen the chance of the patient in his or her resistance to this trypsin toxemia. Many years ago, when I first went over this subject, I rather favored the latter view; but on further study of the problem with larger experience, I am inclined to the opinion that immediate operation would increase rather than diminish the chances of recovery.

Up to the present time I have never had an opportunity really to test this view, because the cases in which I have operated recently have not been of the fulminating type.

CASE REPORT.

CASE II.—Pathol. No. 24143. Acute pancreatitis. Not relieved by removal of stone from, and drainage of, the gall-bladder. Eighteen days later drainage of a peripancreatic cavity filled with cloudy fluid and fat necroses in its wall. Recovery.

This patient was operated upon at St. Agnes Hospital February 7, 1919, by my colleague, Dr. Seegar, who removed a number of soft stones and drained the gall-bladder.

Operative Pathology of First Operation.—Dr. Seegar's notes are as follows: On opening the peritoneal cavity no fluid and no fat necroses were observed. The finding which impressed the surgeon most was a large mass in the region of the pancreas. (The patient was not jaundiced at the time of the operation.) This mass pressed the stomach against the anterior abdominal wall, and the organ was collapsed. The mass felt smooth and was somewhat round or oval in shape. It was present on both sides of the vertebral column; it extended up under the stomach and liver; and it did not pulsate. The right kidney could be palpated and entirely separate from this mass, but the tumor prevented palpation of the left kidney and spleen. It was the size of the head of a fetus. This mass in the region of the pancreas apparently pressed up against the gall-bladder and the cystic duct. (It is important to state here again that the patient was not jaundiced.) The gall-bladder contained a number of soft stones. There is no note on any adhesions about the gall-bladder. The gall-bladder was drained in the usual way and the wound closed.

Clinical History Previous to First Operation.—The patient was a

white female aged 37. She was overweight and very fat. Acute attacks simulating indigestion and gall-bladder colic had taken place at irregular intervals for the past five years, and in some of these attacks there had been jaundice. Between the attacks the patient was apparently in good health. There was usually vomiting during the attacks and the pain radiated to the shoulder.

During the past eight months the attacks had been more severe, of longer duration, and her family physician informed Dr. Seegar that as a rule in these later attacks the patient had been collapsed. The symptoms had at times been so serious that in his opinion myocarditis was present.

The patient entered St. Agnes at the end of a very severe attack in which there was marked collapse in the early hours. Under observation in the Hospital the patient would have an attack of pain, vomit, and the pulse would become very weak,—so much so that on one or two occasions there seemed to be every evidence that she would not recover. It was for this reason that the operation was delayed. Pancreatitis, however, was not considered, but some myocardial lesion. The examination revealed nothing but a mass in the umbilical and lower epigastric area and tenderness and muscle spasm in the region of the gall-bladder. The urine contained no sugar.

When operated upon February 7 by Dr. Seegar, the patient took the anesthetic well as was not shocked.

Post-Operative Notes After First Operation.—In spite of drainage of the gall-bladder, the patient was not relieved of her epigastric discomfort and intermittent attacks of pain, during which the pulse would become very weak. At my examination I could palpate a deep mass between the lower left costal margin and the umbilicus, and this mass was sensitive and the seat of her pain. Palpation on the right side was prevented by the wound. There was no jaundice.

Second Operation, February 25, 1919.—At this time the tube in the gall-bladder had been removed, but the sinus was still discharging clear bile. It was eighteen days since the first operation. The patient took the anesthetic well. The average pulse was 100; blood pressure, 130. The incision was made in the mid-line, above the umbilicus. There was no fat necrosis in the very thick subcutaneous fat, nor in the preperitoneal fat. On opening the peritoneum there was no fluid, but a few disseminated fat necroses could be seen on the surface of the omentum. The omentum was not adherent in this area, nor were there hemorrhages or edema. Lifting out the omentum and transverse colon, I could feel and see an indurated pancreas through the mesocolon extending to its tail. I could also feel a mass apparently on top and surrounding the pancreas, bulging anteriorly above the mesocolon and extending up behind the stomach. The mass was evidently smaller than when Dr. Seegar operated eighteen days previously.

The omentum and colon were replaced in the peritoneal cavity and an opening in the omentum was made in a manner similar to that described in Case I. On making this opening numerous fat necroses

were seen. Deep down in this area I could see and feel a tense membrane, but on this membrane I saw no fat necroses as in Case I. On nicking this membrane, there escaped at least a pint of cloudy fluid containing white granular material (probably necrotic fat tissue). This material resembled that which escaped from the drainage sinus in Case I on the fourteenth day. This fluid was sponged out. The fluid seemed to occupy the lesser peritoneal cavity. What felt like the pancreas, formed its posterior wall. It extended toward the tail in greater depth than toward the head of the pancreas. It extended upward behind the stomach, and it was limited downward by the mesocolon.

It is very interesting to note that the fluid from this cavity which escaped on the exposed tissue produced an almost immediate reaction, so that the tissues stuck together as if they had just been covered with some adhesive substance.

The drainage of the cavity and the closure of the wound were similar to Case I.

Post-Operative Notes After Second Operation.—The patient had no further attacks of acute pain, nor of collapse with a weak pulse. She volunteered almost immediately the information that she was much more comfortable than after the first operation. Nevertheless, the convalescence to complete recovery was much slower than in Case I. There was discomfort in the epigastrium and tenderness in the region of the tail of the pancreas for at least one month. The sinus discharged more purulent material and some pieces of what appeared to be necrotic pancreas. But this tissue was not preserved for microscopic examination. The patient did not leave the Hospital for two months.

REMARKS ON CASE II.

It is quite possible that this is the type of pancreatitis which later, if not operated upon, develops into a peripancreatic cyst and may become of huge dimensions.

SUMMARY OF PERSONALLY OBSERVED CASES.

I find that I have notes upon nine additional cases, making a total of eleven cases between the years 1894 and 1919, a period of twenty-five years. This may be looked upon as evidence that the majority of surgeons have a relatively small experience in the diagnosis and operative treatment of this type of pancreatic lesion.

In reviewing these records, I wish to investigate the following questions:

1. Can pancreatitis be diagnosed clinically?

2. Should operation be performed immediately, even in those fulminating cases first seen in a condition of extreme shock or collapse?

3. What is the best method of exploring the pancreas and of treatment when evidence of acute pancreatitis is found?

CASE REPORT.

CASE III. Pathol. No. 513. Acute pancreatitis. Abscess. Operation. Recovery.

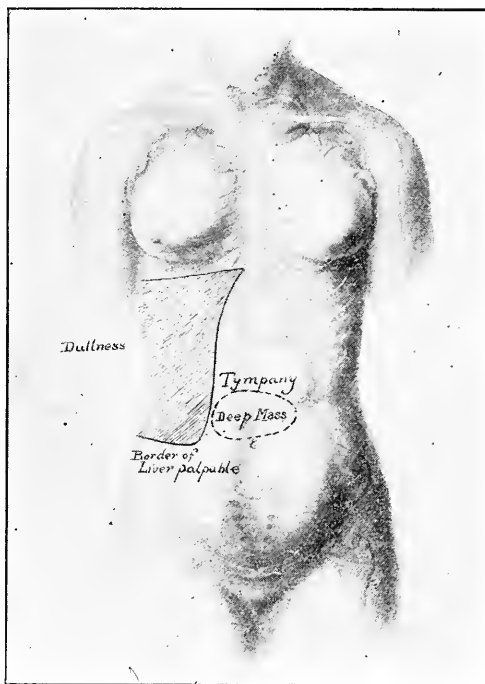


Fig. 1, Case III. Pathol. No. 513. Acute pancreatitis, abscess, tenth day of attack. To illustrate the position of the palpable mass and the enlarged liver.

I assisted Dr. Finney in this operation in 1894. The case was diagnosed clinically by Dr. Thayer and reported by him in the *American Journal of Medical Sciences* in October, 1895. In this instance nothing was done but drainage of the abscess. The gall-bladder and ducts were not exposed at the operation. The patient was well in 1913, nineteen years after operation.

Clinical History.—The patient is a white male, aged 34, overweight and fat, with a history of excess of alcohol, both beer and whiskey. For two years he suffered with morning nausea and intermittent attacks of epigastric pain and vomiting, but no jaundice.

He was admitted to Johns Hopkins Hospital on the tenth day of his first very acute attack, which consisted of epigastric pain, vomiting, fever, distinct toxic delirium, and the examination revealed a mass above the umbilicus. (See Fig. 1.) There was no jaundice. The clinical feature which impressed me most was the evidence of toxemia and delirium. The palpable mass was distinctly in the pancreatic area. The liver was enlarged.

Operative Pathology.—An incision was made in the mid-line above the umbilicus. The liver was enlarged, the stomach adherent to the liver and below the stomach a mass of fat tissue (omentum) studded with yellow, sulphur-like granules (fat necroses). A mass could be felt deep below this omentum behind the stomach, and apparently in the lesser peritoneal cavity. At this time we were not familiar with the fact that pancreatic abscesses are sterile. It was the first case ever observed in the Hospital, and for this reason gauze was packed into the peritoneal cavity between the omentum and the parietal peritoneum.

This operation had no effect upon the fever nor delirium. It relieved the vomiting but not the nausea. A loop of small intestine was coughed out of the wound on the fifth day, and I replaced this with additional gauze drains. On the seventh day Dr. Halsted explored with his finger the exposed omentum studded with fat necroses and opened a large cavity filled with purulent and necrotic tissue. The patient's general condition rapidly improved. During convalescence a number of large pieces of necrotic tissue were discharged from the sinus. These were examined microscopically and showed areas of fat necrosis and the typical stroma of the pancreas. The patient left the Hospital at the end of three months with a small sinus, which healed in a few weeks.

Although this abscess was operated upon on the tenth day, it was really not drained until the seventeenth day, and there is no question that a portion of the pancreas sloughed and was discharged from the wound.

We have no evidence in this case that in the beginning of the attack there was any collapse, nor that it was the fulminating type of pancreatitis. However, the symptoms in the first twenty-four hours were sufficiently characteristic to allow a diagnosis. The later symptoms were fever, delirium and tumor, undoubtedly due to the necrosis of the pancreas and the peri-pancreatic abscess.

Case IV.—Surgical No. 10574. Acute pancreatitis. Abscess. Operation under local anesthesia (Bloodgood) on the twenty-first day of the acute attack. Death twenty-four hours after operation.

This case has been reported by me in the *Johns Hopkins Hospital Bulletin* for January, 1901. The autopsy was reported by Opie in

the *American Journal of Medical Sciences* for January, 1901. The autopsy in this case demonstrated a stone in the diverticulum of Vater similar to a case reported by Halsted and autopsy reported by Opie. But in Halsted's case there was no abscess.

Clinical Note.—The patient was white, male, aged 47. He was not overweight; there was no excess of fat; and there was no history of alcohol. For a number of years there had been attacks called "indigestion." Seven months before admission to Johns Hopkins Hospital there was a severe attack of pain and jaundice lasting three weeks. This was diagnosed gall-stones.

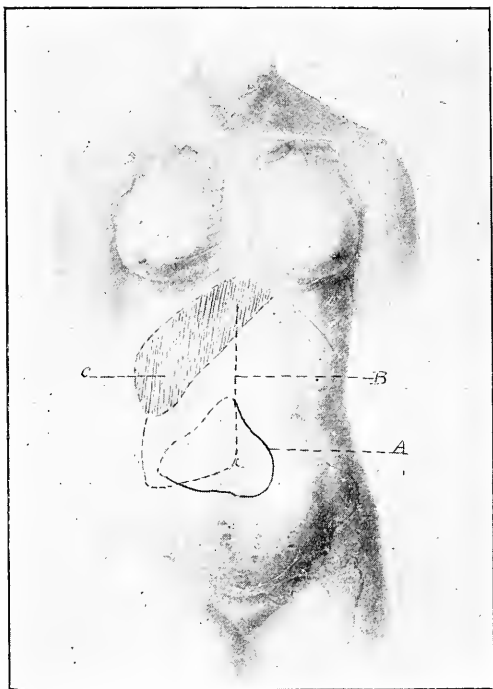


Fig. 2, Case IV. Surg. No. 10574. Acute pancreatitis, abscess; acute symptoms 21 days. A—palpable tumor; B—visible distention; C—liver dullness.

The patient was admitted to the medical side of Johns Hopkins Hospital on the eighteenth day of his last attack. In this attack the onset of pain had been more intense than in the previous attack seven months previously. During the first twenty-four hours there was vomiting, but none since then. On the third day there was fever, and on the seventh day a distinct mass in the epigastrium.

Examination.—This was very similar to that of Case III. The

patient showed delirium and slight jaundice. The leucocytes were 19,000 and the boggy mass (Fig. 2) which could be palpated. The liver was not enlarged as in Case III. The patient was observed on the medical service three days.

When I saw him with Dr. Fitcher I made the diagnosis of pancreatic abscess because of my distinct recollection of Case III.

Operation.—Upon opening the peritoneal cavity the omentum presented itself studded with fat necroses and distinctly edematous. The omentum was adherent by fresh adhesions to the parietal peritoneum. There was no difficulty, by blunt dissection with the finger, in making a hole in the omentum and exposing a second mass covered with fat and fat necroses. This mass was aspirated, and in so doing there were withdrawn a few drops of blood. By blunt dissection, after passing through 3 cm. of fat matted together with an inflammatory exudate and studded with fat necroses, I evacuated a liquid material. The first that flowed out was yellow and gelatinous, and the next brown and thick, like chocolate. The cavity was apparently a large one and had numerous pockets. Cover-slips and cultures showed a bacillus which was not thoroughly worked out.

Autopsy.—The abscess was well drained and there was no peritonitis. The pancreas was very necrotic, and there was a small stone in the diverticulum of Vater.

REMARKS ON CASE IV.

Why did this patient, whose abscess was drained on the twenty-first day, die, and the patient in Case III, whose abscess was drained on the seventeenth day, recover? My notes show that the condition of the patient in Case IV was much more critical. The temperature was 104°, the pulse 130, and the respirations 40.

The diagnosis in this case could have been made in the first few hours of the attack, and most certainly on the seventh day, when a mass could be felt. There is every reason to conclude that an immediate operation would have resulted in recovery.

CASE V.—Pathol. No. 8256. Acute pancreatitis, apparently subsiding. Operation on the fifth day. Recovery.

Clinical Note.—This patient came under my observation at St. Agnes Hospital, in 1907. Up 'til that time I had observed pancreatitis only twice, both abscesses (Case III, in 1894, and Case IV, in 1900) and I was naturally gratified that I made the diagnosis in this case on the fifth day.

The patient was a white male aged 43. He was slightly overweight and fat. There was no history of alcohol. It is very important to note that there was no history of previous attacks nor of infectious diseases nor of trauma.

Before retiring one night feeling well, he drank two glasses of

cold milk and ate a large amount of chocolate candy. Three hours later he was awakened with intense epigastric pain and vomiting. The pain was so intense that he sent for Dr. Kempter, of Chambersburg, Pa., who gave him morphine hypodermatically, and observed him very carefully. At the end of sixteen hours slight jaundice and high colored urine were observed. The vomiting had ceased, the temperature had risen to 102° ; the pain was confined to the epigastrium; and there was distinct rigidity of both upper recti muscles. Dr. Kempter gave the patient no more morphine and nothing by mouth but water. The pain and fever gradually subsided and the jaundice disappeared.

At the end of three and one-half days I saw the patient at St. Agnes Hospital.

Examination.—The pulse varied from 90 to 100, the temperature from 100 to 101° . The patient states that he still has some epigastric discomfort, and in this area there is distinct tenderness and some rigidity. The liver dullness is present and not enlarged; the respirations are abdominal, 26; no mass can be felt, and there is no area of abnormal dullness in the abdomen. The leucocytes were 13,000 with a slight increase in the polymorphonuclears. Although the patient is not restless he looks ill. There is no jaundice and the urine does not give the reaction for bile. The bowels moved by enema. Any food (he was given only liquids) increases the epigastric discomfort.

Diagnosis.—The patient was observed one and one-half days, and my notes made at that time (May, 1907) on the first case of this kind which I had observed read as follows:

The clinical picture suggests acute pancreatitis with recovery, but the possibility of a developing abscess should be considered, and in view of the fact that there are still residual symptoms, it seems the wiser plan to operate.

Operative Pathology.—Although I felt that it was pancreatitis, I first explored the appendix through a small incision at the outer border of the right rectus. On opening the peritoneal cavity there was no fluid, and out of this wound there protruded a small piece of omentum studded with fat necroses. A second incision was made in the mid-line above the umbilicus. There was no fluid; a few fat necroses were found in the omentum, but no hemorrhage and no edema. The stomach, transverse colon and small intestines were not distended. The pancreas was exposed as already described in Cases I and II. As I approached the pancreas the fat necroses increased in number. There was no fluid in the lesser peritoneal cavity. I was not able to see the pancreas as described in Case I, and on palpation the pancreas did not feel abnormal. The gall-bladder was not distended and contained no stones. The drainage in this case consisted of gauze and rubber tissue.

Post-Operative Notes.—The patient claimed that he felt relieved of his epigastric discomfort. There was no discharge, except the serous fluid that one observes after drainage in the peritoneal cavity.

The drains were removed on the thirteenth day, and the patient left the Hospital at the end of four weeks with a very small sinus, which healed rapidly.

Ultimate Result.—In 1916, nine years after operation, the patient wrote that he had had no further attacks and that he was well.

REMARKS ON CASE V.

Apparently this was the type of acute pancreatitis which recovers, and it is a question in this case whether operation had anything to do with the recovery. Apparently in this case and in Case III there was no relation to gall-stones. If there was any necrosis of the pancreas in this Case V, it must have been absorbed, because no necrotic tissue was discharged through the sinus.

CASE VI.—Pathol. No. 8936. Acute pancreatitis with cholecystitis and gall-stones. Operation three days after onset. Death twenty hours after operation associated with complete anuria.

Clinical History.—This patient was referred to me at St. Agnes in 1908 by Drs. Mahle and Holiday. She was critically ill and the examination at once suggested either the fulminating type of acute pancreatitis or general septic peritonitis.

The patient was a white female, aged 45, unmarried. The extreme cyanosis noted by Halsted was present. The pupils were pin-point, probably due to morphine. The respirations were entirely thoracic and gasping. The pulse was very weak and rapid. The leucocyte count was 40,000, the temperature 100°. The abdomen was very much distended, the muscles very rigid, the patient wincing whenever a muscle was pressed upon. On percussion, tympany; no areas of dullness. On catheterizing the bladder there was very little urine. At the end of the third day from the beginning of the attack she was observed by me, and the critical symptoms had then been observed less than twenty-four hours. For the first two days of the attack, according to the physician, the pain was chiefly in the right lower quadrant, and it was looked upon as a slight attack of appendicitis.

The patient was too ill to give any information direct, and both physicians were positive that for two days there had been no very severe symptoms, and that the bowels had moved after enemas until twenty-four hours previously. If that history were correct, it rather suggested a perforated appendix with a general streptococcic peritonitis. The patient was also profoundly under the influence of morphine. There had been no vomiting of fecal character.

Operative Pathology.—The area of the appendix was explored through the outer border of the right rectus. On opening the peritoneal cavity there was no fluid and no signs of peritonitis. The appendix was normal, although the small intestines were quite dis-

tended with gas. On the omentum seen in this exploration there were no fat necroses as noted in Case V. The pelvic organs were normal. On pushing the cecum to the medial side some cloudy fluid was observed which apparently was flowing from the region of the gall-bladder. I then made an upper right rectus incision. The gall-bladder was distended and tense; there were no adhesions; in the space between the gall-bladder, liver, duodenum and meso-transverse colon, there was a serous cloudy fluid. On sponging out this fluid I saw in the mesentery of the transverse colon fat necroses. This is shown in Fig. 3. Apparently this is an unusual place. The pancreas was enlarged. There was no evidence of hemorrhage into the tissues. As the gall-bladder contained stones and very dark bile, the stones were removed and the gall-bladder drained. I also drained the space between the colon, duodenum and gall-bladder, where we found the fluid and majority of fat necroses.

As the patient was critically ill, I had to work very rapidly, but it is my opinion now that it would have been better had I drained the lesser peritoneal cavity through an incision in the gastrocolic omentum as described in Cases I and II.

Sir Berkeley Moynihan, of Leeds, England, was present at this operation, and said that he had seen similar fat necroses in exploring the gall-bladder.

The patient rallied well from the shock of the operation. A rectal tube brought away gas and fecal matter, but catheterization of the bladder found but 30 c.c. of urine.

Previous History in Case VI.—Later I ascertained from the family of the patient and her physicians that for five years she had suffered with intermittent attacks of abdominal pain and vomiting, and that some months previously she had had a very acute attack in which she was prostrated, and that two weeks before her last attack she had been confined to bed for two days.

It was ascertained, also later, that five days before she came into St. Agnes Hospital the patient had had an attack of acute diarrhea, and that in the present attack noted as of three days' duration, the very acute symptoms began forty-eight hours instead of twenty-four hours before her admission to the Hospital.

This retaking of the history demonstrates how difficult it is to get the exact facts and the exact duration of the acute attack. This element of error adds to the difficulty when we try from the study of a number of cases to come to any definite conclusions. But apparently in this case the patient had suffered from sufficiently characteristic attacks to indicate operation some years earlier, and certainly, in this present attack the intervention could have been earlier, before her condition had become critical.

REMARKS ON CASE VI.

This clinical picture corresponds with that form of acute pancreatitis described as fulminating, in which the patient is in shock

or collapse. There is a difference of opinion as to whether or not operation should be done. Personally, I have no evidence that anything is to be gained by delay, but as far as I can make out from my own cases and those in the literature, the chief hope of relief depends upon the recognition of the condition before

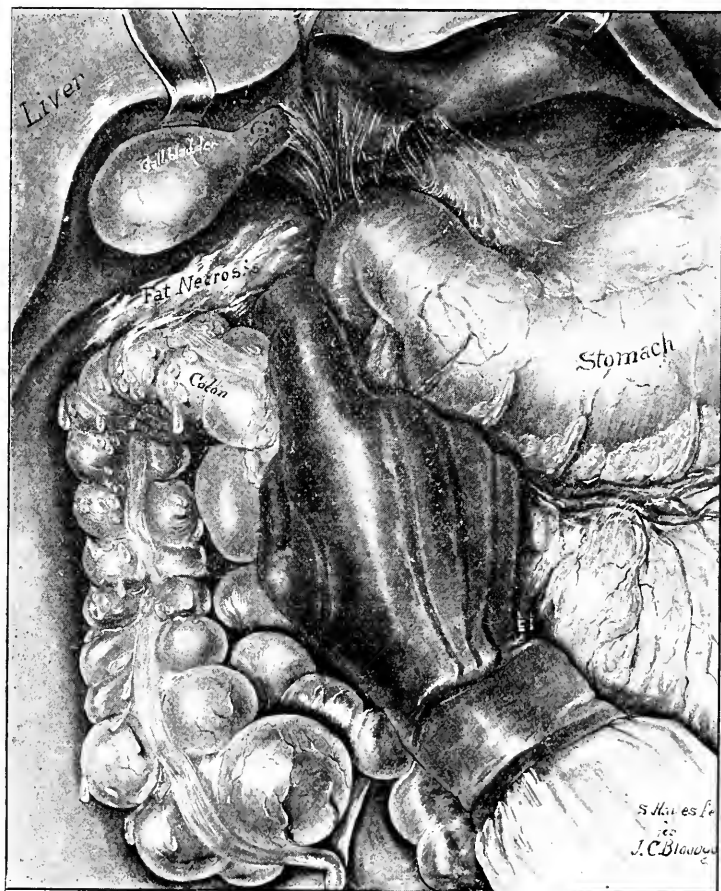


Fig. 3, Case VI. Pathol. No. 8936. Sketch of operative findings showing area of fat necrosis on mesocolon between gall-bladder and duodenum; gall-stones in the cystic duct; adhesions between cystic duct and duodenum.

shock or collapse takes place, and in the majority of cases this seems possible. In Case I, reported in this paper, twelve or twenty-four hours' delay may have found this patient in a condition of collapse.

CASE VII.—Pathol. No. 9900. Acute pancreatitis with definite cyst in the lesser peritoneal cavity and cholecystitis with gall-stones. Operation was done in 1909. Drainage of the cyst in the lesser peritoneal cavity and of the gall-bladder through two incisions. Recovery. Post-operative complication, peripheral neuritis.

Clinical Note.—I was reminded of this case during my operation in Case II, as they were very similar in clinical history and operative findings.

I operated upon this patient in Hagerstown in August, 1909, for Dr. Wertz, and I made the correct clinical diagnosis based upon my previous experience with two cases of pancreatic abscess (Cases III and IV) and a third case operated upon by Dr. Mitchel, in Washington, tissues from which were sent to me with the record.

This patient (Case VII) was a female, aged forty-five; slightly over-weight and with excessive fat. For a number of years there had been intermittent attacks of indigestion without jaundice. The present attack had been more severe and of longer duration than any previous attack. The patient had been ill for at least six weeks. Very quickly after the onset of pain and vomiting there was jaundice and epigastric tumor. The jaundice had disappeared and the tumor had become smaller (compare with Case II). But the patient continued to have epigastric distress, and nausea and vomiting when she took much food. On account of this the diet had been so restricted that she had lost fifty pounds. Despite this she was still over-weight and fat.

This was similar to the clinical picture in Mitchel's case, in which a clinical diagnosis of cancer of the stomach with pyloric obstruction was made.

Examination.—On inspection nothing abnormal was detected about the abdomen. On palpation a deep mass (Fig. 4) was felt in the middle of the epigastrium above the umbilicus, very much as in Fig. 1. The mass was round, the size of two fists. It did not move with respiration. It could be moved from right to left. There was distinct pulsation, which seemed to be transmitted as if the mass rested upon the abdominal aorta. There was not the expansile pulsation of an aneurism. The hand could be placed between the ribs and the mass on both sides. The tumor appeared too suddenly for either a new growth or an aneurism. It followed a distinct attack of gall-bladder colic. As the stomach was empty and there was no muscle spasm, it could be palpated most satisfactorily.

Operative Pathology.—An upper right rectus incision was made. On opening the peritoneal cavity no fluid was found. No fat necroses were present in the omentum. The tumor could immediately be palpated (Fig. 5) through the omentum, and behind the stomach, which was not distended. The stomach was pushed forward and upward, as in Case II. The tumor bulged between the stomach and the transverse colon. On removing the omentum and transverse colon I could easily outline the tumor through the mesocolon. I could palpate the

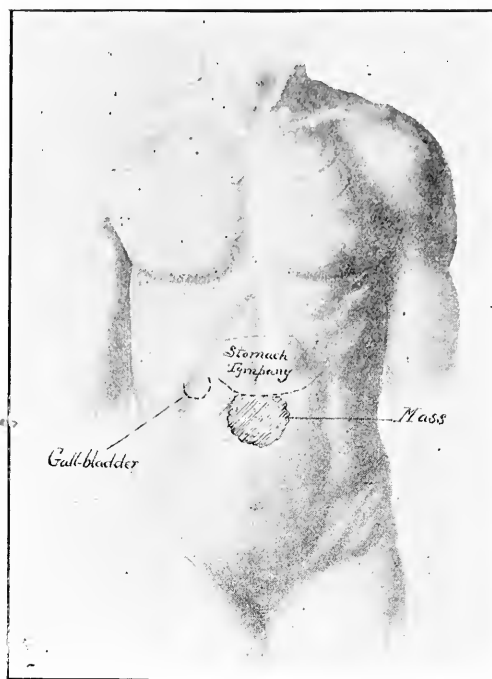


Fig. 4, Case VII. Pathol. No. 9900. Acute pancreatitis with cyst in lesser peritoneal cavity and cholecystitis with gall-stones. Sketch to show the position of the palpable mass and palpable gall-bladder.

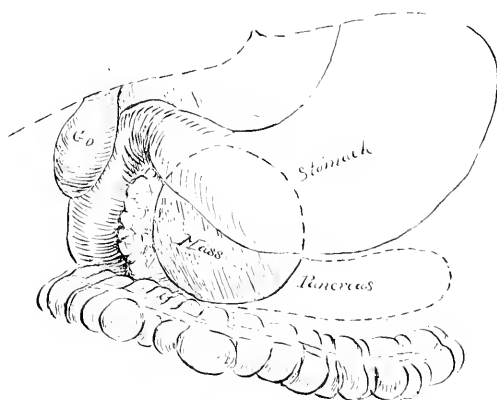


Fig. 5, Case VII. Pathol. No. 9900. Sketch to demonstrate the relation of the cyst in the lesser peritoneal cavity to the stomach and colon. The great omentum has been omitted from the sketch.

tail of the pancreas to the left of the mass and the head of the pancreas to the right, and could demonstrate that the left loin was not the proper place through which to drain this cyst or chronic abscess in the lesser peritoneal cavity. The gall-bladder was filled with stones, but its walls were not thick and it was not distended. The tumor was then exposed through the omentum and gastrocolic ligament, as described in Cases I and II, but through this incision and not through a separate mid-laparotomy. Up to this time no fat necroses had been observed. But in going through this fat I found a few areas of fat necrosis, but these were distinctly different in color from those observed in the more acute stage of pancreatitis, being a very pale yellow and of more crystalline appearance. On passing through the gastrocolic omentum I exposed a distinct cyst wall, elicited fluctuation, excluded expansile pulsation, and on aspiration obtained clear fluid. On incising this cyst there was first a clear fluid, and then distinct chocolate colored fluid containing globules of fat (see Case IV). This material was not present in Cases II and III. The cyst contained about five ounces of this material. As soon as we had sponged out the fluid the cyst collapsed. I could not see the pancreas, nor did palpation demonstrate that it was very much indurated. The cavity was drained with a tube and iodoformed gauze similar to Cases I and II. The gall-bladder was opened, emptied and drained with a tube. The bile was black and viscid. A stone was also removed from the cystic duct which was at first overlooked.

The closure of this wound with two distinct drainage tubes surrounded by gauze was complicated, and I am confident that for drainage of the lesser peritoneal cavity the mid-line incision, as described in Cases I and II, is preferable.

Postoperative Notes.—I saw this patient again on the ninth day. The gall-bladder tube was draining well, but the drainage of the lesser peritoneal cavity was not satisfactory. For two days there had been vomiting and fever with rapid pulse. I removed the drainage and washed out from the cavity purulent material, necrotic fat and what appeared to be necrotic pancreatic tissue. This gave almost immediate relief.

One month later the patient was admitted to St. Agnes Hospital with the definite clinical picture of multiple neuritis associated with a mental syndrome. The diagnosis was confirmed by my associate, Dr. Herring. These symptoms began about the fourteenth day. As far as I can make out the healing of the drained cavity did about as well as in Cases I, II and III in this paper. It is the only case of multiple peripheral neuritis that I have observed after operation. The patient was very slow in getting well and has never fully recovered. But she has had no further attacks suggestive of gall-stone or pancreatitis, and no sugar in the urine.

CASE VIII.—Pathol. No. 9856. Apparently a case of recurrent acute pancreatitis associated with chronic pancreatitis. First operation was cholecystostomy. (No jaundice at that time). Second opera-

tion was drainage of the common duct. (Jaundice at that time and fat necroses were found at operation.)

Clinical Note.—This patient was referred to me by Dr. Fletcher, at Johns Hopkins Hospital, in 1909. He was a white male, aged 61, of apparently good health, slightly overweight and very fond of good living, with a moderate amount of alcohol. For a number of years this patient had suffered with intermittent attacks of epigastric colic and glycosuria. Apparently each attack was definitely associated with overindulgence in food and alcohol. He was admitted to the Johns Hopkins Hospital at the end of the third week of a rather severe attack from which he had not completely recovered. There had been jaundice in that attack which had practically disappeared. The examination revealed sensitiveness and muscle spasm in the region of the gall-bladder. There was neither fever nor leucocytosis. The clinical picture was one of cholecystitis, and a definite relation of the attacks to food, and the intermittent glycosuria suggested also a chronic pancreatitis.

Operative Pathology.—On opening the peritoneal cavity no fluid and no fat necroses were seen in a very large omentum. The gall-bladder was distended, the walls thick, and the omentum and mesocolon were adherent to the gall-bladder. The latter contained no stones, but a great deal of white gelatinous material, which microscopically proved to be hypertrophied Luschka's glands. The cystic, common and hepatic ducts were distinctly thickened by an inflammatory reaction, which was also present in the wall of the duodenum, and the pancreas as far as it could be palpated was indurated. But I could find no fat necroses. We had, therefore, a definite picture of an inflammatory process involving the head of the pancreas, the duodenum slightly, and the gall-bladder and ducts. This condition must have been subsiding, because the jaundice at operation had disappeared and the fever which had been present in the early days of the attack had subsided.

Drainage of the gall-bladder completely relieved the patient's symptoms. The sugar had disappeared from the urine before operation.

Second Attack.—I warned this patient against any overindulgence in food or alcohol. He was readmitted to the Johns Hopkins Hospital September 28th, about two months after the first operation. For the first month after operation he was free from attacks, but then he returned to hard work, and on three occasions broke his diet at banquets, which occasions were immediately followed by definite attacks. The first, one month previously, consisted of a sharp attack of epigastric pain lasting for two hours. The second attack, two weeks previously, was characterized by more pain, fever and jaundice, lasting a few days. The third attack, one week before readmission, was most severe in epigastric pain, in chills with temperature rising to 103° , and with definite jaundice. The patient was observed for two days. Although the skin was still jaundiced, bile had returned in the stool and had almost disappeared from the urine.

Although the temperature did not rise above 100.5° , the leucocytes remained at 22,000.

Operative Pathology at Second Operation.—No fluid was found in the peritoneal cavity. More adhesions were seen than at first operation, which were easily separated, bringing into view the gall-bladder, which was not much distended, the cystic duct, the common duct, which was greatly distended (not distended at the first operation), and the pancreas, which was much more indurated than at the first operation. Over the head of the pancreas there were distinct disseminated fat necroses. On opening the common duct there were no stones. A probe could easily be passed into the duodenum. Both the common duct and the gall-bladder were drained.

Result.—This patient recovered and had no further attacks up to 1917, eight years later.

Because we did not find fat necroses at the first operation is no evidence that they were not present in the lesser peritoneal cavity. There is no question that they were present at the second operation. One might conclude from a case of this kind that drainage of the common duct is more effective in this type of pancreatitis with obstruction to the common duct, than drainage of the gall-bladder. There is apparently no question that at the first operation we observed a distinct pathological picture of cholangitis and cholecystitis associated with pancreatitis, but the inflammatory lesion was subsiding, and there also appears to be no question that at the second operation the chief lesion was a pancreatitis, but that the swelling of the head of the pancreas which had blocked the common duct was subsiding.

CASE IX.—Pathol. No. 14039. Acute pancreatitis. Clinically and at operation diagnosed intestinal obstruction due to cancer or diverticulitis of the sigmoid. This patient died six days after operation (colostomy of the cecum) and an autopsy demonstrated a necrotic pancreas with fat necroses.

Clinical Note.—I saw this patient with Dr. Cassidy on April 14, 1913, twenty-four hours after the onset of what the patient and his family insisted was the first attack; and after a most careful history and examination made an incorrect diagnosis and operated four hours later and did not recognize, on opening the abdomen, the lesion that had produced the symptoms. In this case surgical history repeated itself, because in the majority of cases of acute pancreatitis the diagnosis of intestinal obstruction had been made.

This patient was a white male, aged 62. Before this attack his health had always been good. However, he was overweight, very fat, of sedentary habits and a huge eater; but he did not use alcohol to excess. This first attack began a few hours after eating peanuts and ham sandwiches. There was first intense abdominal pain in the epigastrium. The patient then took a number of doses of cathartics, which increased the pain, but did not produce vomiting nor a stool. When seen by Dr. Cassidy, in about the eighth hour of the attack, he was suffering intensely with pain in the epigastrium, but Dr.

Cassidy found no muscle spasm. Morphine gave temporary relief of pain, but the patient again took cathartics without instructions from his physician, which not only increased the pain, but produced vomiting. This was twelve hours after the beginning of the symptoms, and for the next twelve hours pain and vomiting were characteristic symptoms.

Examination.—Twenty-four hours after the onset the patient was vomiting bile-stained material. He looked slightly cyanotic and toxic. The temperature was 104°; the respirations (thoracic) were 30; and the pulse 100. The patient had just taken an enema, and during the examination had a sudden abdominal colic different from the epigastric pain, which was quickly followed by a foul-smelling, liquid stool. The examination at St. Agnus Hospital, about two hours later, found the leucocytes to be 23,000. Gastric lavage brought away bile-stained fluid. An enema was ineffectual. The cyanotic and toxic appearance had increased; the distension was more marked; all the abdominal muscles were slightly rigid; the liver dullness was not obliterated, and there was no dullness in the flanks.

Diagnosis.—My notes state that we considered chiefly sigmoiditis with partial obstruction, and decided to explore in the region of the cecum for a temporary colostomy. As I review this case, I remember distinctly that I did not think of pancreatitis, and I cannot conceive now why I did not make a diagnosis of pancreatitis. The symptoms were far too acute and developed too rapidly to be associated with obstruction of the colon.

Operative Pathology.—On opening the peritoneal cavity cloudy fluid was encountered. Had the fluid been blood-stained or brown, I should have at once suspected pancreatitis. But this fluid is not always present, especially in early cases. On the omentum there were no fat necroses, which were fortunately present in the omentum in Case V. But experience has led us to know that we cannot always depend upon finding fat necroses, especially in early cases, outside of the lesser peritoneal cavity. The small intestines were more distended than the large. This should have made me suspicious of my diagnosis of obstruction of the sigmoid. When I explored the sigmoid from the right wound, I got the impression that its surrounding fat was indurated. But every surgeon knows the difficulty of diagnosis by palpation, when the patient has a huge omentum, fat appendices epiploicae, and large amounts of fat in the mesocolon. The appendix was removed and the cecum was sutured to the parietal peritoneum for later opening.

Postoperative Notes.—The symptoms were not relieved. There were hiccough and vomiting of fecal character, not relieved by gastric lavage. Thirty-six hours after operation the cecum was opened, and this was followed by the discharge of huge quantities of fecal material. Following this there was great improvement in the general and abdominal condition of the patient. Vomiting ceased; the hic-

cough; however, although better, persisted. The distention almost disappeared.

For two days the patient was apparently improving. Then there was a sudden change for the worse: hiccough without vomiting (lavage showed no gastric retention), diminished urine, delirium and rapid pulse. Death.

Autopsy.—A partial autopsy was made by the resident, Dr. Loos, who found fat necroses in the omentum between the stomach and colon, hemorrhages, bloody fluid in the lesser peritoneal cavity; a huge pancreas covered with fat necroses, and containing areas of pancreatic necroses. There were no gall-stones (there had been no jaundice). The microscopic examination of the tissue removed confirmed the diagnosis of fat necroses and areas of necrosis in the pancreas.

REMARKS ON CASE IX.

In view of this patient's condition at the time of the operation, twenty-eight hours after the onset of the attack, and the number of days that he lived after the operation (six days), I am inclined to the opinion that if the correct diagnosis had been made and the lesser peritoneal cavity drained, as in Cases I and II, the patient would have recovered.

The evidence of obstruction of the colon as revealed at the operation was not sufficient. I should have explored the upper abdomen through a second incision.

CASE X.—Pathol. No. 18229. Acute pancreatitis with cholecystitis and gall-stones in the gall-bladder. Operation seven days after the beginning of the attack and twenty-four hours after the beginning of the symptoms with collapse. Drainage of the lesser peritoneal cavity and gall-bladder. Recovery.

Clinical Notes.—This case cannot be considered one of fulminating pancreatitis, but the patient was critically ill and was getting worse. I made the correct diagnosis in this case.

The patient was a white male, aged fifty, admitted to Johns Hopkins Hospital September 15, 1915. The suggestive etiological factors were typhoid fever eleven years previously for the gall-stones; overweight, obesity and overindulgence in food for the pancreatitis. There were definite previous attacks—first, for a few years, of belching after meals; then, for eighteen months, attacks of epigastric pain and belching beginning two hours after meals and disappearing five hours after eating. Often in these attacks the patient would induce vomiting to relieve the pain. The attacks were at first once a month; then twice a month. Seven months previously there was a very acute attack with jaundice lasting five days. These attacks all suggest gall-stones.

The present attack began seven days ago. At the onset the pain

was so intense that he showed signs of collapse. The most marked feature has been that anything taken by mouth not only gave discomfort, but was as a rule not retained. If the patient took no food nor water, he had less discomfort and no vomiting. Despite an almost starvation diet, the patient had a second attack of intense pain, with collapse twenty-four hours before admission, and jaundice appeared for the first time.

Examination.—The patient complained chiefly of pain in the epigastrium, more marked on the left side, but the intensity of the pain had abated. Even small quantities of water produced vomiting. The vomitus was bile-stained. The jaundice was disappearing, and enemas brought away brown fecal matter. The pulse varied from 100 to 116; blood pressure, 126; respirations, 24; and the leucocytes were 22,000. The abdomen was moderately distended in the upper half; the upper halves of the recti muscles were rigid; sensitiveness was slight. The patient looked toxic, but there was no cyanosis.

The most marked symptoms were regurgitation of any food, muscle rigidity, and pain in the epigastrium.

Operative Pathology.—A diagnosis of pancreatitis and gall-stones was made. A right rectus incision to explore the gall-bladder revealed brown-stained fluid in the peritoneal cavity. A distended, thick-walled gall-bladder contained stones. In separating adhesions between the gall-bladder and duodenum fat necroses were found in the fat over the head of the pancreas and in the parietal peritoneum. A second incision was made in the mid-line above the umbilicus. There were fat necroses in the omentum. On opening the lesser peritoneal cavity through the gastrocolic omentum there was evacuated a brownish fluid which microscopically contained blood and chemically bile and pancreatic ferments. The pancreas was not seen, but was felt to be enlarged and indurated. The lesser peritoneal cavity was drained as noted in Case I; the stones were removed; and the gall-bladder was drained. The closure of the wound was similar to Cases I and II. It is interesting to note that the staphylococcus albus was found in the fluid from the gall-bladder, but the fluid in the lesser peritoneal cavity was sterile.

Postoperative Notes.—The patient remained in the Hospital forty-one days. For some days epigastric discomfort and hiccough persisted. From the lesser peritoneal cavity there was discharge not only of purulent material, but of pieces of necrotic pancreatic tissue.

The subcutaneous fat in both wounds became necrotic, but the silver wire sutures through-and-through prevented the gaping of the wound. For at least fifteen days the drainage from the gall-bladder and the lesser peritoneal cavity were not perfect, but after that time drainage was apparently perfectly established and the patient's discomfort disappeared and his general condition improved.

Ultimate Result.—It is now (June, 1919) almost four years since the operation, the patient is well and has suffered from no recurrent attacks.

CASE XI.—Pathol. No. 20724. Acute pancreatitis with jaundice; one stone in the gall-bladder. Operation forty-eight hours after the onset of acute symptoms. Drainage of the lesser peritoneal cavity and gall-bladder. Recovery.

Clinical Note.—This patient was referred by Dr. Slade, of Reisters-town, Md., on December 19, 1916, forty-eight hours after the beginning of an acute attack. The diagnosis of acute pancreatitis was made and operation performed at once.

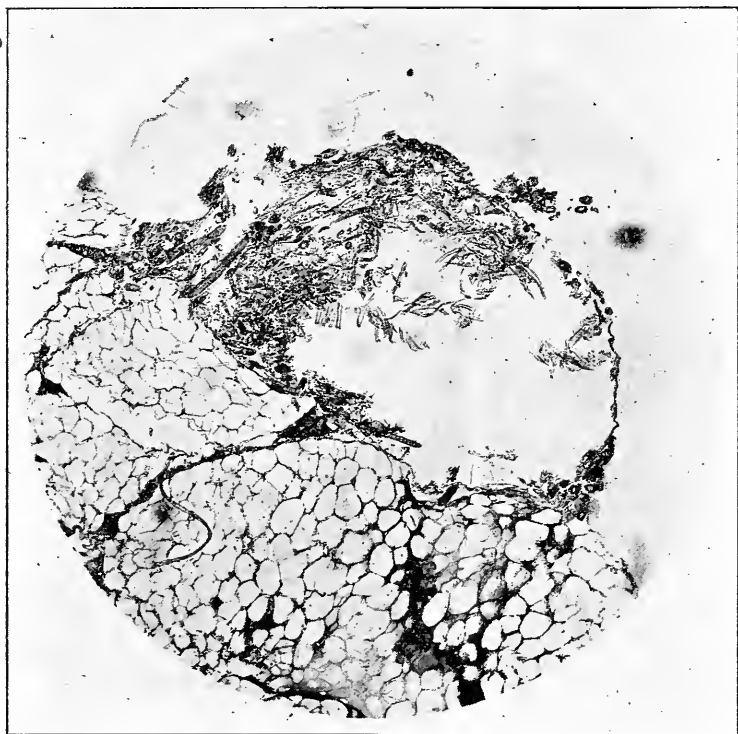


Fig. 6, Case XI. Pathol. No. 20724. Photomicrograph (low power) of area of fat necrosis in omentum.

The patient was a white female, aged forty; overweight and obese. In 1908, eight years previously, I had operated upon her for chronic lactation mastitis, but at that time we were not suspicious of any gall-bladder lesion. The patient, however, had then suffered from attacks of indigestion, but had paid no attention to them. Four months before operation she was admitted to St. Agnes Hospital under the care of my colleague, Dr. Freeman, who made the diagnosis of chronic cholecystitis and advised operation, which was refused.

At that time the patient was suffering with intermittent attacks of epigastric pain and vomiting, especially after the morning meal.

The attack which preceded the operation was more acute than any previous attack. There was intense pain in the epigastrium and in the gall-bladder area. The pain was referred to the shoulder. Vomiting of all food; jaundice.

Examination.—Temperature, 101°; leucocytes, 16,000; and blood pressure, 144. The pulse varied from 68 to 102. Slight jaundice, but some bile in stools. Definite distention in the epigastrium, muscle spasm and sensitiveness with definite tenderness and muscle spasm in the gall-bladder area.

Operative Pathology.—A right rectus incision for exploration of the gall-bladder. Clear fluid was found in the peritoneal cavity. A few fat necroses were seen on the omentum. The gall-bladder was not distended. There were adhesions. One stone was present in the gall-bladder; no stone in the duct. The head of the pancreas was indurated. A second incision was made in the mid-line. Fat necroses were seen in the omentum. On opening the lesser peritoneal cavity there escaped blood-stained fluid and more fat necroses were found in that region. The pancreas was not seen, but felt very much indurated. Drainage as in Cases I and II.

Postoperative Notes.—Rather rapid recovery. No discharge of necrotic pancreatic tissue from the lesser peritoneal cavity. June, 1916, the patient was two and one-half years well.

Microscopic Appearance of Fat Necroses.—From this, Case XI, I removed a small area of fat necrosis and Fig. 6 illustrates the microscopic appearance.

CONCLUSIONS.

Can pancreatitis be diagnosed clinically? Yes. The characteristic symptom is pain in the epigastrium present on both sides. If there is vomiting, the pain is not relieved by it. The most suggestive sign on examination is the sensitiveness in the epigastrium and the rigidity of the upper portion of both recti.

When the patient is carefully questioned, he will admit a distinct feeling of prostration all out of proportion to the duration of the pain and rarely, if ever, observed in an equally severe pain of the gall-bladder or kidney.

The signs of prostration may not be evident in the pulse rate and blood pressure. Fever and leucocytosis appear to be late symptoms.

PREVIOUS ATTACKS.

In the majority of cases there will be a history of previous attacks of one or two types; epigastric pain and distinct gall-

bladder colic. When the latter is present, gall-stones or cholecystitis are found at operation.

A history of jaundice in previous attacks, or evidence of jaundice at the examination, can not be looked upon as characteristic of pancreatitis, nor do they exclude pancreatitis.

The great majority of these patients show not only overweight, but various degrees of obesity. They are all of sedentary habits, and with hardly an exception are over-eaters. Overindulgence in alcohol is distinctly a factor of less importance than overindulgence in food.

CLINICAL DIAGNOSIS.

This is essential, because the evidence of pancreatitis may not be found until the lesser peritoneal cavity is opened. (See Case I.)

RELATION TO GALL-STONES AND CHOLECYSTITIS.

In the literature this relationship is rather overemphasized. The majority of surgeons—perhaps all—will operate upon from 100 to 300 cases of gall-stones or cholecystitis and meet only one case of acute pancreatitis in which drainage of the lesser peritoneal cavity is essential for recovery.

There seems no doubt that there is a definite relationship between pancreatitis and gall-stones, with or without cholecystitis, and cholangitis, and Opie's explanation of regurgitation of bile may explain some but not all the cases. Apparently we have not as yet exact knowledge as to the etiological factor or factors, and we must for the present attempt to make the diagnosis and relieve the condition without this exact knowledge of the etiology.

THE CORRECT ATTITUDE OF THE FAMILY AND PHYSICIAN IN THE PRESENCE OF ACUTE ABDOMINAL SYMPTOMS.

The public should be instructed that when there is an acute attack of abdominal pain with or without nausea, the best treatment is absolute rest in bed and nothing by mouth, either solid or liquid, and that a physician should be sent for at once.

Both the public and the medical profession should be repeatedly warned against the administration of any cathartic in the early stages of an acute abdominal attack. Ochsner has correctly stated

this matter. If a cathartic is needed, no harm will ensue by its delay; if it is not needed, it is dangerous.

Even in this small list of cases, the patient most critically ill had been given huge doses of cathartics, while the patients in better condition had had no cathartics and no food.

MORPHINE.

These patients are in such pain that morphine must be given for their relief, but in my experience an abdominal colic not relieved by one hypodermic of morphine should be regarded with suspicion of a lesion requiring immediate operation, and the patient should be at once transported to a hospital, so that an operation can be done at once, if further examination indicates it.

In acute abdominal lesions enemata and gastric lavage are helpful in making the patient more comfortable, in preparing them for operation, should it follow, and are often of diagnostic value.

The careful examination of these patients in the first few hours of the attack, and the obtaining of a correct history from the patient or family, should in the majority of cases lead to a correct diagnosis, at least to the point of sending the patient to the hospital, where one with larger experience will be able to make the diagnosis as to whether operation is indicated or not, and this patient will be in far better condition not only from the fact of earlier intervention, but because the acute lesion has not been irritated by cathartics or food, or obscured by an overdose of morphine.

DIET.

As the vast majority of patients who come under observation in the critical stage of acute pancreatitis have been warned by previous attacks, the public and the general profession should be instructed on the importance of paying more attention to vague symptoms of indigestion and to slight attacks of epigastric pain and belching or vomiting, with or without a transient jaundice, and the most important treatment is not drugs, but restriction of diet, a most thorough examination, and, if possible, an operation before the acute attack takes place.

It is quite possible that pancreatitis without accompanying gall-stones can be relieved by proper diet and reduction of overweight and obesity.

I know of no definite finding in a thorough examination which will allow a diagnosis of pancreatitis unless the patient is seen in an attack of epigastric pain and rigidity. The diagnosis must be made more by exclusion and from the history of the attacks.

SHOULD OPERATION BE PERFORMED IMMEDIATELY, EVEN IN THE
FULMINATING CASES FIRST SEEN IN A CONDITION OF
EXTREME SHOCK OR COLLAPSE.

The evidence strongly suggests that death in acute pancreatitis, with or without abscess, is due to a toxemia produced by the pancreatic ferments which in some as yet unexplained way gain entrance to the lymphatic or blood circulation. There is no question about the death and the autopsy findings. Apparently there is no question that recovery takes place in animals in which pancreatitis has been produced experimentally and in the human being with all the clinical evidence of the disease. From my own experience and the reading of the literature I am impressed with the conclusion that drainage of the lesser peritoneal cavity through the gastrocolic omentum meets the indications more than any other suggested treatment.

At this operation the gall-bladder with rare exceptions should be explored and drained if indicated. Perhaps we may learn that in jaundiced cases the common duct should be drained.

My own experience leads me to conclude that the so-called fulminating type of pancreatitis is not common, and that our failures to cure have been due to an improper operation in the early cases or to late intervention. My Case VI. is an example of an incomplete operation in a relatively early case, and in Case IX. the death of the patient was undoubtedly due to an incorrect diagnosis, while Cases I., X. and XI. seem to show the efficacy of a correct diagnosis and correct treatment of acute pancreatitis in the early stage.

We must repeat here that pre-operative diagnosis is essential. Exploratory laparotomy does not always reveal the blood-stained peritoneal exudate and the fat necroses. Even the exploration of the head of the pancreas when the area of the gall-bladder is explored may not disclose a pancreatitis. The best method of examining the pancreas, if one does not wish to go through the gastrocolic omentum and fat without more evident signs, is to

withdraw the omentum and colon and inspect and palpate the pancreas through the mesocolon.

WHAT IS THE BEST METHOD OF EXPLORING THE PANCREAS AND OF
TREATMENT WHEN THERE IS DISTINCT CLINICAL EVIDENCE
OF PANCREATITIS, OR SIGNS AT THE EXPLORATORY
LAPAROTOMY?

This question has already been answered in the preceding discussion.

SOME PRACTICAL ASPECTS OF THE PHYSIOLOGY
OF DIGESTION.

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To a "rookie" in the service, the insignia of the Army Medical Corps is an inspiration to supreme endeavor, as it has been consecrated by the spirit and the achievements of some of the greatest men in medicine. Beaumont, the Army Surgeon, laid the foundation for the physiology of gastric digestion, even though he was stationed at a small army fort in the wilderness of Michigan. The modern development of cerebral localization dates back to the Franco-Prussian War of 1870 and the battlefield observations of Dr. Fritz. We owe the conquest of malaria to Dr. Ross, of the Indian Medical Service; Reed, Carroll, Agramonte and Lazear gave us victory over yellow fever; and Gorgas virtually built the Panama Canal by the practical prevention of these two maladies. Other names might be added; but surely Beaumont, Reed, Gorgas, have added lustre to the medical caduceus and have set a standard of service challenging supreme effort on our part, and the present conflict is sure to bring exceptional opportunities to the Army Surgeon alert and prepared to grasp them.

During the past five years the physiological laboratory of the University of Chicago has been fortunate in having at its service a "second Alexis St. Martin," a healthy man with a gastrostomy and complete cicatricial stenosis of the esophagus of twenty years' standing. The stricture of the esophagus followed alkali corrosion, the stomach not being noticeably injured. The gastrostomy is large enough to admit a rubber tube three-fourths inch in diameter, and a tube of this size is always kept in the fistula. This permits the introduction of ordinary solid foods well masticated. The man chews his food in the ordinary way, spits it out into a bowl and puts it into the stomach with a syringe. Liquids are put into the stomach in the same way with or without first putting part into the mouth.

You need not be told that this case furnishes an unusual oppor-

tunity of investigating some of the problems of gastro-intestinal functions in health and disease that have developed since the days of Dr. Beaumont and the first Alexis St. Martin. The fistula is large enough to permit direct inspection of the interior of the stomach. It permits the introduction of balloons, rubber tubes, small electric lights, electrodes, etc., for various lines of research. It permits the collection of pure gastric juice while the man is masticating his dinner, etc.

We have four methods of objective study of gastro-intestinal physiology and pathology in the living individual:

1. The unusual and well-known case of Alexis St. Martin led to experimental gastrostomy in dogs and finally to the isolated stomach pouch and the classic work of Pawlow on gastric juice and gastric digestion.

2. The stomach tube, and later the duodenal tube, has contributed data of importance to fundamental medicine. But the old "garden hose" type of stomach tube should be replaced by the more physiological tube recently introduced by Dr. Rehfuess, as the latter tube can be *swallowed* by 90 per cent. of patients with a little coaxing, and can be kept in place for hours without gastric or subjective disturbances.

3. The X-ray method has not only cleared up fundamental questions of gastro-intestinal motility, but it is (especially the fluoroscope) an indispensable adjunct to all gastro-intestinal diagnosis.

4. The balloon method was applied to the analysis of gastro-intestinal functions years ago, but this method has not yet been put to full use in the clinics. As developed in the Chicago Laboratory the method is applicable to patients of all ages (including the prematurely born infant); and it is indispensable in the analysis of the motility and sensations of the empty stomach; it is a necessary adjunct to the X-ray in studying the motility of the filled stomach, as it reveals some phenomena not disclosed by the X-ray; and, finally, the method is indispensable in the analysis of all types of sensory disorders of the gut, such as anorexia, bulimia, nausea, gastralgias, vagotonia, and the pains of gastric and duodenal ulcers.

The details of the method are given in the monograph on the "Control of Hunger in Health and Disease," University of Chicago Press, 1916, and we need not take time to go into it here,

except to say that it is applicable to all cases that permit of the passage of a stomach tube or catheter at least three-eighths of an inch in diameter. It requires *patience*, and some knowledge of physiological technique, and fundamental physiology. These may be considered defects in the method, but in the way of compensation it yields important information not disclosed by the other methods of analysis.

Using this fistula case as a general guide, and extending the work to normal persons, to various types of patients, and to experimental animals, we have secured new data on human gastric juice, its secretion and chemistry in health and disease, its rôle in peptic ulcer, etc. We have been able to throw some light on the nature of hunger in ordinary life and in prolonged starvation; on the anorexia of fevers, on bulimia, on the polyphagia of diabetes, on the pains of peptic ulcer, on the control of the pylorus, on the toxemias of intestinal obstruction, on the sensibility of the gut, etc. Making full use of an unusual opportunity, we have been able to supplement and in some cases profoundly modify some of the current views of gastro-intestinal physiology and pathology.

With your permission we will review a few of these new facts and interpretations.

I. THE MOTOR PHENOMENA OF DIGESTION.

The motor phenomena of the alimentary canal in the healthy man are admirably correlated with the particular processes of digestion and absorption which take place in the different regions of the gut. We need but instance the rapid process of deglutition; the tonic grip of the cardia preventing regurgitation into the esophagus; the tonic pressure of the gastric fundus on the food mass, coupled with the antrum peristalsis moving the gradually digesting surface layer of the food towards the pylorus; the rhythmic relaxations and contractions of the pyloric sphincter; the forward movement (peristalsis) and the segmentation movements of the small intestine, where the main digestion and absorption takes place; the antiperistalsis of the ascending and transverse colon, permitting partial drying of the waste; the haustral movements of the descending colon leading to defecation; the tonic grip of the anal sphincter, etc., anyone moderately familiar with the various phases of secretion, digestion and absorption,

recognizes at once the nice correlation of type and rate of motion with digestion and absorption. But this recognition does not explain the "how" of the correlation, nor its breakdown in disease.

(a) *Mastication*.—The various types of movements exhibited by the different regions of the gut are primarily under local and partly under central nervous system control, mainly reflex. Man possesses some direct or voluntary control over the two ends of the alimentary canal, viz., the mouth and the rectum. That is, over the processes of mastication and defecation. The voluntary control of the latter process is, as you all know, an indirect one through habit formation and abdominal pressure. We have heard a great deal in recent years of the hygienic importance of eating slowly and masticating thoroughly. These modern views are essentially correct, although the healthy and vigorous individual has such a large factor of safety in digestive power, that he can bolt his food without evident injury. In our crusade for Fletcherizing let us not forget that regularity of waste elimination is of much greater importance to health and physical efficiency than careful mastication of the food.

Thorough mastication presupposes palatable food. Unpalatable food, if eaten at all, is usually bolted so that the disgust may be over with as soon as possible. Thorough mastication of unpalatable food presupposes a degree of stoicism, fanaticism, or a sense of duty not found in the average man; let us not forget this fact in our endeavor to improve the army rations and the army mess. Palatability is the surest means to thorough mastication. Given palatable food, thorough mastication yields the following desirable results:

1. Facilitation of digestion by the more complete division of the food material.
2. Increased secretion of appetite gastric juice owing to the more prolonged stimulation of the taste organs in the mouth, and by the increased dilution of the food with the water of the saliva.
3. A more rapid and complete development of the sense of satiety.
4. Decreased food consumption, owing partly to the more rapidly developing sense of satiety and partly to the sense of fatigue of the muscles of mastication.

5. More complete sterilization of the food by the acid gastric juice.

In normal individuals these are desirable effects. Only in exceptional cases will increase in gastric digestion, or increase in gastric juice secretion, lead to undesirable results, in the form of hypersecretion or too rapid evacuation of the stomach. The latter may induce bulimia and excessive ingestion of food.

(b) *Movements of the Stomach in Digestion.*—The motor phenomena of the stomach and intestines during digestion are now fairly well understood, thanks to the fluoroscope and X-ray protography, supplemented by the balloon method. We have the peristaltic movements of the antrum, and the gradual tonic contraction of the fundus of the stomach. This tonic contraction of the fundus or body of the stomach is not a steadily increased tension, but appears as tonus waves of from one to three minutes' duration. These tonus waves of the fundus increase in intensity as the stomach gradually empties, and when the stomach is empty they pass into the so-called hunger contractions of the empty organ.

The behavior or control of the pyloric sphincter is more variable and less well understood. Cannon's researches on cats led him to believe that a certain degree of acidity on the stomach side favors the opening of the pylorus, while the acidity on the duodenal side causes a reflex contraction of the pylorus. In man, however, there are so many exceptions to this acidity control, especially from the stomach side, that we may fairly question whether it is to be regarded as a general law. We have found that in the normal man and in animals there is a correlation between the opening of the pylorus and the tonus rhythm of the fundus of the stomach, in such a way that the pylorus opens at the height or near the end of each tonus wave. We do not know as yet to what extent this is modified by various disease conditions, such as "peptic" ulcer or gastric neurosis, but we do know it obtains in the healthy individual. This co-ordination is relatively independent of chemical reaction in the stomach. It is quite clear from our work that a certain degree of acidity or a certain degree of alkalinity or complete neutrality on the stomach side is compatible with normal pyloric rhythm and gastric evacuation in the normal person.

Under all conditions so far investigated, both in man and

animals acid acting on the duodenal side of the pylorus never fails to introduce reflex contraction of this sphincter. This is true even after cutting all the extrinsic nerves to the pylorus. It is, therefore, partly a local mechanism. It is an interesting fact that in some species (cat) we may section and re-suture the stomach close to the pylorus, or we may similarly section the duodenum close to the pyloric sphincter so that both local as well as extrinsic nerves are eliminated; in either case pyloric control returns after a time so that we have normal evacuations of the stomach. It would thus appear that the pyloric sphincter may be controlled either from the duodenal side or from the stomach side, at least in some mammals. In the case of the empty stomach the degree of pyloric tonus is variable even in health. Water, egg white, and other liquid foods, may start to pass into the intestine immediately on entering the stomach. It is said that this is invariably the case in nursing infants. In man resection of a considerable part of the body of the stomach is said to lead to persistent patency of the pylorus (Perthes). The other extreme, or pyloric spasm, is, of course, more serious, and its etiology equally obscure, except those associated with duodenal ulcers where we in all probability are dealing with an exaggeration of the normal reflex closure evoked from the duodenal mucosa.

Regurgitation of duodenal contents into the stomach appears to be a normal physiological process. This regurgitation is favored by high acidity, or the presence of fat, in the stomach. The regurgitation may serve a useful purpose by introducing pancreatic enzymes, thus aiding retarded gastric digestion, or neutralizing a too high gastric acidity.

(c) *Contractions of the Empty Stomach.*—By the term empty stomach we understand a stomach empty or nearly empty of food. Strictly speaking, the stomach is never completely empty, as there is usually some gastric juice, saliva, or duodenal contents present (25-50 c.c.). It was generally held, up to a few years ago, that the empty stomach is atonic and flacid and exhibits no contractions. Hence the practice of prescribing fasting to insure motor and secretory rest of the gut in certain types of gastro-intestinal disorders. We now know, thanks to special methods of investigation on both man and animals, that in healthy individuals the empty stomach is never completely at rest, and that, on the whole, it exhibits a greater degree of tonus and

motility than the filled stomach. This is true for all species of vertebrates so far investigated down to and including the frog.

The special method of investigation is the introduction of a small balloon into the stomach, the balloon being connected with a rubber tube through the esophagus to a recording manometer which registers the variations in gastric tonus and contractions on the kymograph. This method may be combined with that of fluoroscope and X-ray photography. This is done by covering one balloon with a bismuth or barium paste and slipping another balloon over it so that, after being introduced into the stomach, it will give the bismuth shadow without any material other than the smooth rubber balloon touching the stomach. The contractions of the empty stomach come in periods, varying from fifteen minutes to one hour, and in rare instances two and one-half hours. These periods are characterized by gradual increase in tonus and on these tonus waves are superimposed a series of more rapid contractions each lasting from eighteen to thirty seconds. These individual contractions become gradually stronger and may end in an incomplete tetanus at the end of the period, this tetanus lasting from one or two minutes up to fifteen minutes. That is to say, the stomach in this condition stays maximally contracted, but the contraction is not absolutely continuous. This is then followed by more or less abrupt relaxation of the gastric tonus and a period of relative quiescence lasting for a half hour to several hours. But even during this period of quiescence the body of the stomach exhibits feeble tonus rhythm, at least in the case of the healthy individual. The contractions of the empty stomach start at the cardia and sweep over the entire stomach in the form of peristaltic waves, except in rare instances, when the stomach appears to contract simultaneously throughout its whole extent. These contractions of the empty stomach are more vigorous and continuous in the new-born infant than in the adult. They are present in the prematurely born infant, and probably go on *in utero*. They persist with normal or increased activity in prolonged fasting (man at least twenty days; dog practically up to the point of death from starvation). The contractions decrease in frequency and vigor in old age.

There are usually very active contractions of the small intestine during the periods of contraction of the empty stomach. At first it was thought that there is a distinct difference between the

digestion contractions of the full and the hunger contractions of the empty stomach, because of the different ways that local or external conditions effect these movements. More careful investigation, however, shows that, while the digestion contractions start near the transverse band and involve primarily the antrum, as the stomach empties, the digestion contractions start farther and farther up the body of the stomach until the origin is definitely at or near the cardia when the stomach is nearly or completely empty of food. That is to say, the digestion contractions fuse imperceptibly into the hunger contractions and apparently the only difference between the two is (1) the greater vigor of the empty stomach; (2) the involvement of the entire stomach in the contractions; and (3) the different effect of the contractions on consciousness.

(d) *The Local and Reflex Nervous Control of Gastro-Intestinal Movements.*—It is well known from experiments on animals, and probably true in the main for man, that complete section of all the extrinsic nerves to the alimentary tract does not permanently impair or modify the motor phenomena of that part of the gut which is made up of smooth musculature and the so-called Auerbach's plexus. But clinical observations seem to indicate that the destruction of the extrinsic nerves to the large bowel or destruction of the lumbar and sacral cord in man, produces greater disturbances (paralysis) in the movement of the large intestine than similar injuries induce in the dog. In general, we may say that the primary motor control of the greater part of the gut lies in local neuro-muscular mechanism. *The Auerbach's plexus must be regarded as the central nervous system for the gut*, just as the brain and the spinal cord is the central nervous system for the reflex control of the skeletal system.

We find that after elimination of all the extrinsic nerves, local reflexes involving distant segments of the gut, may still be obtained by stimulation of the gastro-intestinal mucosa; stimulation of the intestinal mucosa by mechanical or chemical means still causes a reflex inhibition of the stomach movement. This reflex inhibition is not as strong nor as complete as when the extrinsic nerves are intact, but it is still there. In the same way stimulation of the gastric mucosa causes inhibition of the stomach motility after the section of both vagi and all the splanchnic nerves. Irritation of the mucosa of the large intes-

tine does not inhibit the movements of the stomach after section of the extrinsic nerves to the large bowel.

The extrinsic nervous system of the gut involves both motor and inhibitory effects. The motor path to the upper end of the gut, *i. e.*, the esophagus, cardia, the body of the stomach and the pyloric sphincter, come through the vagi nerves. The motor nerve fibres to the other end of the gut, *i. e.*, the colon and the rectum, come from the lumbar and sacral cord through the nervi-irigentes, and the hypogastric plexus. Thus we see that the main motor innervation of the gut emerges from the two extreme ends of the spinal cord, while the inhibitory nerves to the stomach and intestines emerge from the thoracic lumbar cord via the splanchnic nerves. This separation in motor and inhibitory fibres is not complete, as the vagi contain some inhibitory fibres to the cardia and possibly some to the stomach, and the splanchnic may contain a few inhibitory fibres to the intestine as well as to the stomach. In the main, however, this general scheme holds true for men and the lower animals.

Stimulation of the peripheral end of the vagi nerves induces contractions of the small intestines, but it is not known whether this means that the motor fibres in the vagi extend direct to the small intestine throughout its course. Some neurologists and physiologists assume that this is the case. If this is the correct interpretation we would have nerve fibres in some species thirty to forty feet in length.

The important question, however, is not the anatomical course of these fibres, but how they are controlled in health and disease. The first important generalization, from work on man and animals, is the fact that *there is no known method of reflexly or voluntarily stimulating or augmenting the motor nervous mechanism through the vagi in the healthy individual*. All stimulations, external and internal, painful, pleasant or indifferent, if they connect with the extrinsic nerves efferent of the gut at all, produce inhibition, *i. e.*, they throw the inhibitory mechanism into activity rather than the motor mechanism. Thus, any and all stimuli, acting in the mouth; any and all stimuli acting on the mucous membrane of the stomach; all stimuli sufficiently strong, such as heat or cold, or pain, acting on the skin, cause inhibition of gastric motility, both the digestion contractions and the hunger contraction. All strong emotions, so far studied, cause similar

inhibition. The only instance where we have succeeded in obtaining reflexly increased activity of the motor mechanism through the vagi, is by previous section of the splanchnic nerves on both sides and the stimulation of the central end of the gastric branches of one vagus, leaving the other vagus intact. Under such conditions we obtain a reflex augmentation of gastric tonus and contractions. It is evident that there are sensory fibres distributed in the stomach capable of increasing gastric tonus reflexly through the medulla or mid-brain in the absence of the splanchnic inhibitory mechanism, but in the normal animal, or man, with the inhibitory mechanism unimpaired, this motor reflex is apparently completely nullified by inhibition.

The control of vagus tonus is a very important problem, both to the physiologist and the clinician because this factor is probably involved in cardia spasm, in pyloric spasm, in various spasms of the body of the stomach, in the gastric crisis of tabes, and in the various states included in the term vagotonis. We have some evidence that the tonus and the motility of the empty stomach is increased in the gastric crisis of tabes. The tonus of the vagus motor mechanism is increased during sleep, probably through a relative depression of the inhibitory nervous mechanism.

II. THE SENSIBILITY OF THE ALIMENTARY CANAL.

There is a great deal of confusion on this subject in the minds of physiologists and clinicians owing in part to lack of accurate knowledge and in part to lack of clear definitions. Thus, some people, in speaking of the sensibility of the stomach, or of the alimentary tract in general, refer only to nervous impulses that may influence consciousness. It is obvious that there may be important sensory components from the gut which play solely on the reflex or subconscious processes. The sensations or conscious states that we ordinarily associate with, or refer to the alimentary tract in normal persons, are:

1. Hunger.
2. Appetite.
3. Satiety.
4. Fullness.
5. Defecation urge.
6. Peculiar sensations of heat that may follow strong chemical stimulation of the gastro-intestinal mucosa.

In persons with gastro-intestinal disorders we may have:

1. Nausea.
2. Anorexia.
3. Bulimia.
4. Pains of "peptic" ulcer.
5. Pains of gastralgias.
6. Pains of gastro-intestinal colic or cramps, gas pains.
7. An ill defined and not definitely localized general discomfort or tension referred to the viscera frequently experienced in mild gastritis, enteritis and constipation.

All these refer to sensory nerves and afferent impulses that influence consciousness. In addition, we have sensory nerves in the alimentary canal whose stimulation results in:

1. Vascular reflexes.
2. Respiratory reflexes.
3. Alteration of general reflex excitability of the spinal cord and the lower brain center and reflexes involving some of the glands, the salivary, the gastric, the adrenals, etc.

The mucosa of the alimentary tract seems to be entirely devoid of the tactile sensibility characteristic of the skin and the mucous membrane of the mouth. There is some temperature sensibility in the esophageal mucosa and in the mucosa of the anal canal. The question whether the gastric mucosa is provided with temperature nerve-endings has long been a matter of dispute. We are all familiar with the fact that hot foods or hot liquids produce a feeling of warmth in the stomach, but people who deny temperature sensibility of the gastric mucosa have explained those sensations as referred from the esophagus or to stimulation of the nerve-endings of the skin of the abdomen by the diffusion of heat through the abdominal wall. It can be now stated as a definitely established fact that the gastric mucosa has temperature nerves, both for heat and cold, but this sense is much less delicate than in the case of the skin. These statements are based upon direct experiments or on normal persons adequately controlled. Whether or not the intestinal mucosa has temperature nerve-endings is an open question, but if so they are evidently less numerous or a lower grade than those in the esophagus or stomach. In other words, there is a decrease in the temperature sensibility of the gastro-intestinal mucosa as we proceed from the oral and anal openings inward.

The normal gastro-intestinal mucosa appears to be entirely devoid of *pain nerve-endings*. The mucosa may be cut or crushed in various ways in a conscious person without producing pain.

The peculiar sensation of warmth or heat that arises from the stomach on taking strong acids or concentrated alcohol, or similar irritating substances, may be due to the stimulation of the heat nerve-endings in the mucosa, but weaker chemical stimuli, acting in the stomach, produce an effect on consciousness similar to, if not identical with, the sensation of *appetite*. This sensation is produced, even when the substance, such as a glass of cold water or a glass of very weak alcohol, is put into the stomach through a tube without touching the mouth or the esophagus. It is thus evident that a certain moderate chemical stimulation of the gastric mucosa contributes to the element of appetite. This is a pleasant sensation and various substances in the food may be adequate stimuli under normal conditions. We ordinarily ignore this gastric component of appetite because the appetite induced by tasting, seeing or smelling palatable food is very much more powerful; hence, the weaker factor is not recognized when food is eaten in the ordinary way.

Hunger.—Hunger has been explained in various ways, but until recently relatively little accurate work has been directed towards analysis of the various factors involved, although *hunger is as primitive and as important to the race as the urge of sex*. Hunger has been ascribed to a direct stimulation of a hypothetical hunger center in the brain by the lack of food substances in the blood. It has been ascribed to stimulation of sensory nerves in the organs in general by a lack of food substances in the blood or in the tissue cells. It has been ascribed to stimulation of nerves in the stomach by an accumulation of gastric juice in the secreting glands. It has been ascribed to the stimulation of nerves in the mucous membrane of the stomach. *And finally it has been shown to be due, at least in its important components, to a type of contractions of the empty or nearly empty stomach*. Biologists who ascribe the sensation of hunger to lack of food substances in the blood or in the tissues, forget the fact that hunger arises before intestinal digestion and absorption is completed and hence, while the blood and the tissues are flooded with nutrient substances. They have also forgotten that hunger, at least as ordinarily experienced, is periodic. It comes and goes with periods of intense activity separated by periods of relative quiescence.

While there is no evidence of a similar periodicity in accumulation or scarcity of blood and tissue nutrients. The suggestion that hunger is produced by stimulation of the sensory nerves in the gastric mucosa, owing to the folds of the empty stomach rubbing against one another, dates back at least 150 years to the great physiologist Haller. Since the days of Haller we have had much speculation and little work in this field. It has now been shown that when the stomach is empty or nearly empty the contractions of the empty organ we just described give rise to the pangs of hunger at least in the normal person. In the sick individual or in the individual suffering from various types of neurosis, or in prolonged starvation, these contractions may give rise to other effects on consciousness, such as the feeling of sick stomach, feeling of nausea or the feeling of general epigastric distress, but in the normal individual the contractions give rise to the varied degrees of pressure, pain and emptiness that we associate with or call *hunger*. We have shown that it is the gastric contractions that give rise to the hunger sensation and not the central sensation of consciousness of hunger that induces the gastric contractions. The empty or nearly empty stomach appears to be the essential element in producing the hunger pangs. Contractions of the lower end of the esophagus and of the intestine may also contribute, but certainly these factors are of subsidiary importance.

The sensations produced by these contractions are due to stimulation of nerve-endings in the muscular coats of the organ, *i. e.*, hunger is a type of kinesthetic or muscle sense. The very strong hunger pangs become very uncomfortable or painful, just as the strong or prolonged contractions in the skeletal muscle may give rise to the pains of contracture or fatigue.

The problem of control of hunger is, therefore, a problem of the control of the emptying of the stomach and control of the contraction of the empty or nearly empty stomach, provided the sensory path of the vagi and the central paths and brain centers remain normal. Disturbances anywhere, either in gastric motility, in vagi conductions or in the parts of the nervous system concerned with the elaboration of the conscious sensations of hunger will upset the mechanism.

We have been for a number of years endeavoring to work out the normal and practical control of hunger in man and animals.

In the present emergency this may not seem a practical problem, as the world query today is not how we may increase hunger but how we can appease it. We have found that the hunger contractions decrease with age, being maximum in early life or shortly after birth. They persist or are even increased in prolonged starvation, both in man and animals, although in the later stages of starvation mental depression and other central changes may lead to alterations in the conscious effects of the gastric contractions. The view that hunger disappears after three or four days' fast is an error. The gastric hunger contractions in man persist with undiminished vigor, if not with increased vigor, at least during the first twenty days of complete starvation. This is a matter of direct demonstration. The individuals may not feel them as definite or strong hunger, but rather as nausea or epigastric distress after the first few days.

The hunger contractions are increased in diabetes, clinical and experimental. They are decreased in most fevers, clinical and experimental, including thermic fever or heat prostration. The depression or failure of the hunger contractions in fever is not due to a direct action of bacterial toxins on the gastric motor mechanism. In general, the presence of the hunger contractions or the absence of the hunger contractions parallel the call for food or the refusal of food on the part of the man or the experimental animal. To a certain extent we may say that the hunger mechanism is under nervous control in that the motor fibres through the vagi and the inhibitory fibres through the splanchnics, control the contractions of the empty, as well as the filled stomach. We do not yet know whether the periodicity of the hunger *i. e.*, of the hunger contractions, are due to periodic variations in the tonus of the vagi or the tonus of the inhibitory sympathetic mechanism, but it is clear that the hunger mechanism is primarily automatic and independent of the central nervous system as the contractions persist with no material modifications after the isolation of the stomach from the central nervous system.

We have also given some attention to the question of chemical control. We have been able to show that there is something in the blood of the starving animal or of the diabetic animal that induces hunger contractions of the empty stomach. The nature of this substance (or substances) is a matter of conjecture. In diabetes one thinks naturally of the acetone bodies, but experi-

ments with these gave negative results. So far as we can sum up the situation at present the *hunger mechanism is a primary gastric automatism, not directly related to the immediate needs of the tissues for nutrient material*. By heredity the stomach in health acts as automatically and almost as periodically as the beating heart. When food is introduced in the stomach the contractions are diminished and modified so that they do not influence consciousness. When the stomach is empty or nearly empty or when, for any reason, these very strong contractions appear even in the well-filled stomach, the nerve impulses produce a disagreeable tension or painful pressure effect on consciousness that we desire to alleviate by partaking of food. This is the primary sensation of hunger.

There appears at present no certain way of increasing hunger except by increasing the body metabolism through physical work or external cold. The various stomachics or bitters that have been used in medicine to improve hunger from time immemorial, have no effect on the hunger mechanism when given in therapeutic doses into the stomach. When given by mouth they inhibit or depress the hunger mechanism in proportion to the bitter taste or strong stimulation of the nerve-endings in the mouth.

Associated with the hunger pangs we have several subsidiary phenomena such as nervousness, headache, feeling of weakness and dizziness and in some cases even fainting. We have shown that these elements are also largely of reflex origin involving stimulation of nerves in the gastric muscularis by the strong contractions of the empty stomach. They are evidently not due to any lack of nutritional material in the blood or tissues because they disappear temporarily by filling the stomach with non-nutritional material and when food is taken they disappear practically with the first few mouthfuls, *i. e.*, before there can be any appreciable digestion. The feeling of weakness, developed in prolonged fasting, is more permanent and due, in part, to tissue starvation. It is likely that the continued tonic contractions of the abdominal muscles also contribute to the feeling of emptiness which is experienced particularly in prolonged starvation.

Appetite.—Appetite is ordinarily confused with hunger, appetite being regarded as mild hunger and hunger defined as strong appetite. This view appears to be entirely fallacious, as appetite is an entirely pleasant state of consciousness associated with our

memories of food, particularly the taste, the smell, and the sight of foods. Appetite may exist without hunger and is apparently a state of consciousness depending on individual experience. It is, therefore, modified by experience and we can gradually acquire, particularly in early life, appetite or taste for the most varied food substances. It is well known to all of you that appetite is no certain guide to the nutritious value or general wholesomeness of food substances, especially when modified by modern processes of manufacture in way of separation, concentration or degradation of the natural foods. It is a point of practical importance in the present emergency to remind ourselves and to impress on the civil population, as well as the men in the ranks, that the taste or appetite for wholesome food materials, not hitherto in general use, is gradually acquired by persistent use. That our mess officers and cooks must endeavor to render the food attractive and palatable is self-evident. But, provided the diet is well balanced and varied, the ingredients digestible and otherwise wholesome, let every man realize that lack of the particular taste and flavor imparted to the food by mother's or grandmother's cooking is a non-essential.

Satiety and the *sense of fullness* depend partly on a certain degree of distension of the stomach and abdomen and also on the degree of palatability of the meal and the duration of stimulation of the taste and organs of the mouth; thus, the quantity of food being the same, satiety satisfaction is more complete when a meal is eaten slowly than when it is bolted.

In persons with various gastro-intestinal disturbances we meet sensory complexes from the alimentary tract of the greatest practical importance. As you all know, *nausea* can be produced by over-stimulation of the sensory nerves in the gastric and intestinal mucosa, or, given gastritis or enteritis, even such a normal weak stimulation as water at body temperature may produce nausea. Nausea of gastro-intestinal origin, except that induced by absorption of toxic substances, is purely a question of intensity and duration of stimulation of the sensory nerves, usually of the mucosa, but in some cases of the muscularis.

The nature of *anorexia* is more obscure. Unquestionably we may have anorexia purely of central origin. The anorexia of fevers is associated with absence or depression of the gastric hunger contractions. This is also true of the anorexia experi-

enced in gastritis or enteritis. In these conditions gastric hunger contractions may return before we have a return of hunger and appetite. That is to say, in certain stages of gastro-enteritis the gastric hunger contractions give rise to a sensation of nausea or sick stomach rather than to that of true hunger.

Bulimia, or excessive hunger, has not been extensively studied by objective methods. We have had one case of what appeared to be true bulimia, *i. e.*, an excessive discomfort from the hunger contractions of the empty stomach and an absence of true satiety even when the stomach was filled with food up to a point of marked distress. In this individual the gastric hunger contractions were usually not stronger than normal and there was no evidence of an unusual excitability of the gastric sensory nerves. The bulimia in this case, therefore, pointed to a disturbance in the central brain mechanism; but any condition of too rapid emptying of the stomach will probably lead to various degrees of bulimia, since this leads to the too rapid or frequent appearance of gastric hunger contractions.

The pains of "*peptic ulcer*" is another factor of greatest practical importance. Most gastro-enterologists at present appear to accept a view that the ulcer pains are due to acid irritation of nerves and nerve-endings in the ulcer area. This view is at the basis of the alkali treatment and the frequent feedings of the ulcer patient. It has long been known that in most cases the ulcer pains are periodic and the pains have been termed hunger pangs by some clinicians. *It has now been shown that the periodic ulcer pains occur synchronally with the strong contractions of the empty or nearly empty stomach, and are primarily excessively painful hunger contractions.* The excessive painfulness of these contractions does not appear to be due to the contractions being stronger than normally, but rather to a state of hyperexcitability of the sensory nerves. However, *in periods of marked exacerbation of the ulcer syndrome the gastric contractions during digestion, ordinarily not felt at all by the normal person, produce pain in the ulcer patient.* We all know that the digestion contractions increase in vigor as gastric digestion advances and this is partly the reason why ulcer pains come one-half hour, one hour, or two and one-half hours after eating. It is primarily a question of gastro-intestinal contraction, not of gastric acidity.

There is some evidence, particularly in cases of duodenal ulcers,

that contractions of the duodenum itself and prolonged spasms of the pyloric sphincter contribute to the ulcer pains. At the end of a hunger period the ulcer patient may experience ulcer pains when there are no marked contractions in the stomach; or during the period of gastric hunger contractions; the pains may be felt at the end of each contraction rather than at the height of the contractions. Now we have already pointed out that these tonus contractions of the body of the stomach, *i. e.*, the hunger contractions, are associated with the opening of the pylorus and hence with passage of the acid gastric contents into the duodenum. Given excessive acid gastric contents or hyper-excitability of the duodenal-pyloric reflexes, each hunger contraction will lead to strong tonic contractions of the duodenum and reflex spasm of the pylorus, and these types of contractions unquestionably produce varying degrees of pain. There is no direct evidence that the pains of peptic ulcer, duodenal or gastric, is a chemical pain in the sense that the pain is produced by acid irritation, in the absence of contractions. Nevertheless, we see that acidity may indirectly contribute to the pains by modifying or intensifying certain contractions of the duodenum and the pylorus. Alkalies will, of course, diminish or temporarily control this pain factor; so will ingestion of water or foods, and we now know that the gastric factor of the ulcer pains may be temporarily controlled even by giving acids, as acids in the stomach cause a temporary inhibition of the stomach contraction.

The pain of colic, cramps, and tenesmus and flatulence, are evidently also contraction pains. All gastro-intestinal pains of definite peripheral origin are, therefore, essentially contraction pains, wherever they arise in the gut—contraction pains due to stimulation of nerve-endings in the muscular coat of the gut itself.

The pains of gastro-intestinal origin have a profound effect on the subconscious life and on the lower reflex centers; and the same appears to be true of excessive sensory impulses from the alimentary tract not definitely felt as pain. This includes vascular and respiratory reflexes and the reflexes involving the salivary and possibly the adrenal glands. It is a noteworthy fact that stimulation of the sensory nerves of the large bowel produces as marked effect on the individual as the stimulation of the central end of the sciatic nerve. It is very curious that the surgeon can maintain an absolute inexcitability of the gut in

the face of these easily demonstrated facts. It seems clear that the importance of the sensory component from the alimentary tract in normal life, as well as in certain groups of diseases, is at present not sufficiently realized, either in practice or research.

III. THE SECRETION OF HUMAN GASTRIC JUICE.

(a) *The Continuous Secretion of Gastric Juice.*—The continuous secretion of gastric juice in the absence of food in the alimentary tract, and in the absence of cerebral processes relating to appetite, is a well-known phenomenon in certain types of gastric disorders, but it is generally assumed by physiologists that in the absence of psychic stimulation the gastric glands cease to secrete almost as soon as the stomach is empty of food and that the glands remain practically quiescent up to the next feeding. This quiescence is supposed to be sufficiently complete to render the surface of the stomach neutral or alkaline between meals, as a rule. This view is based essentially on the observations of Beaumont, on the famous Alexis St. Martin and on the classic work of Pawlow on dogs.

This view is certainly an error. We have seen that in health the empty stomach is never completely at rest as regards motor function. The gastric glands in the healthy individual are also continuously active in the total absence of food or obvious nervous excitatory factors. The continued secretion varies from a few c.c. up to 150 c.c. per hour. This is true gastric juice and not a residue from the liquid part of the previous meal, swallowed saliva, or regurgitated duodenal contents, as all these factors can be eliminated by proper control. In the Chicago laboratory this continuous secretion has been studied in two gastric fistula cases, normal persons except having the esophagus completely closed owing to alkali corrosion. In one of these, a healthy man, now 34 years old, the gastrostomy and the complete stricture of the esophagus is of 27 years' standing. The observations have been extended to so many normal individuals, young, adult, and old people, that we are convinced that we are dealing here with a general phenomenon.

The average rate of the continuous secretion does not exceed 30 to 60 c.c. per hour, but in a number of individuals it may reach as high as 100 or 150 c.c. per hour without being associated with any gastro-intestinal disorders usually ascribed to

gastric hypersecretion. We are, therefore, inclined to believe that clinical hypersecretion is a symptom and not the cause of the gastric disorders usually found in cases of so-called hypersecretion. It is an interesting fact that when the continuous secretion is removed from the stomach every ten or fifteen minutes, the total secretion per hour is greater than if removed only every thirty or sixty minutes. Evidently some of the gastric juice is passed continuously into the duodenum.

What is the significance of this continuous secretion? It is evidently not an adaptation to immediate needs, as there is no food to be digested. It seems to us that Pawlow has overemphasized the principle of nice adaptation of the quality and quantity of the digestive juices to the quality and quantity of the food. It is obvious that the continuous secretion initiates gastric digestion in cases of eating without appetite, in cases of forced feeding, in cases of ingestion of unpalatable food, or in cases of feeding of the unconscious person by means of the stomach tube. The continuous secretion is rich in pepsin, is usually lower in acid than the appetite gastric juice, as the percentage of acidity is essentially a question of secretion rate. The gastric juice produced by a more rapid continuous secretion may approach an acidity equal to that of the gastric juice secreted on feeding.

We have not yet succeeded in explaining the mechanism involved in the continuous secretion. It may be normal vagotonia. It may represent absorption of secretive hormones from the small intestine and the large intestine, as these parts of the gut practically always contain food in various stages of digestion, or bacteria acting on debris from the mucosa itself. It may be due to the absorption of secretagogues developed by the auto-digestion of gastric juice. Gastric juice contains proteins that are digested in the stomach by pepsin hydrochloric acid. In all probability this yields secretagogues similar to those produced by pepsin hydrochloric acid hydrolysis of the food proteins. The continuous secretion is not markedly increased in prolonged starvation. It is decreased and may be entirely absent in fevers, and in various types of gastritis.

(b) *The Appetite Gastric Secretion.*—This depends on the taste, sight or smell of food on the different nerve paths to the brain, on the existence of the cerebral state of appetite, and, lastly, on the different secretory fibres in the vagi nerves to the

gastric glands. The elucidation of the mechanism and importance of this gastric juice secretion we owe mainly to the Russian physiologist Pawlow. The most important sensory path is that of the taste nerves. In some people, smell and sight of food causes little or no secretion. It is an interesting fact, as recently brought out by Dr. Taylor, of the Mayo Clinic, that appetite gastric juice secretion is absent in the newly born and the young infant. Evidently appetite secretion depends on the presence of appetite sensation or memory, and since this is a matter of individual experience, it cannot be or is not inherited.

The rate of secretion of gastric juice in normal adults on masticating palatable food, varies from one and one-half c.c. to twelve c.c. per minute, the average rate being about three and one-half c.c. per minute. The secretion commences practically as soon as we start to masticate the palatable food and it begins to decline practically as soon as the taste of the food has disappeared from the mouth on the cessation of the meal. The appetite secretion is greatly diminished or absent in gastritis or any inflammatory condition of the gastric mucosa, in fevers and in strong emotions of anger or pain.

What is the significance of this appetite gastric juice? Following Pawlow, it has been generally held that appetite gastric juice is a necessary initiator of gastric digestion, and hence the indigestion or retarding of digestion on eating unpalatable food or on eating palatable foods without appetite. This view is erroneous, as the following facts will convince you.

1. According to Taylor, there is no appetite secretion in normal infants and yet these infants suffer no gastric indigestion.

2. After double vagotomy in dogs and cats there is only a temporary retardation of gastric digestion. After a few days the gastric digestion and evacuation return to normal, despite the fact that there can be no longer secretion of appetite gastric juice.

3. In the experimental animal forced feeding of unpalatable food does not appreciably delay gastric digestion.

4. We know that gastric juice itself is not an absolute requirement for adequate digestion and absorption of foods because these processes evidently can go on to a degree meeting the nutrient requirements in the individual in the so-called achylia gastrica cases, hereditary or acquired, and in individuals with the entire stomach

removed. In fact, in the achylia cases the stomach, as a rule, empties itself of food more quickly than in normal individuals.

5. We have for weeks at a time removed all the appetite secretion of gastric juice from the stomach of the gastric fistula case (Mr. V.) before the food was put into the stomach through the fistula, without producing gastric indigestion or gastro-intestinal disorders.

Of course, the appetite gastric juice is a useful factor when present. This cannot be denied. But if the gut is otherwise normal, especially in regard to motor functions and the continuous gastric secretion, absence of the appetite gastric juice is of no serious matter. The presence of appetite and appetite gastric juice is, therefore, of importance mainly as an index that the alimentary canal is capable and ready to receive and handle the food.

We have made extensive studies on the action of various stomachics or bitters on the appetite gastric secretion, both in man and dogs. The action of these medicines is insignificant if not entirely negative. The appetite gastric secretion is not appreciably increased. In some instances the ingestion of food appears to be slightly increased, but we do not know whether this slight increase in food intake signifies an actual improvement in metabolism.

(c) *Hormone or Secretagogue Secretion of Gastric Juice.*—As you know, protein food in contact with pepsin hydrochloric acid in the stomach, appears to yield a substance which when absorbed acts as specific stimulus to the gastric glands. This constitutes practically an automatic mechanism correlating gastric juice secretion with the sojourn of protein food in the stomach. A substance ("gastrin") has been isolated by a number of workers from the gastric mucosa and other organs which, when injected intravenously, or hypodermically, causes a profuse secretion of apparently normal gastric juice through an action directly on the gastric gland cells. When this substance ("gastrin") is given by mouth it has no effect and is, therefore, useless as a therapeutic agent in achylia or dyspeptic conditions.

We have recently made some observations on gastric juice secretion in fevers, experimental and clinical. All three types of secretion—the continuous, the psychic and the hormone secretion—are depressed or completely abolished by fevers of suffi-

ciently high temperature. This appears to be a direct action on the gland cells, because the injection of "gastrin," which is known to act directly on the gland cells, fails to cause secretion of gastric juice in fevers. In what way fevers paralyze the gastric glands must be determined by future investigations.

IV. CHEMISTRY OF HUMAN GASTRIC JUICE.

On the subject of chemistry of gastric juice we shall only touch on two or three of the more practical aspects.

(a) *Acidity*.—Human gastric juice, secreted as a result of masticating palatable food, without being mixed with saliva or duodenal contents, has an average total of acidity of .45 to .55 of one per cent. with a free acidity of from .4 to .5 of one per cent. When the juice is secreted at the slow rate the acidity is lower. You will note, gentlemen, that this acidity is equal to 120 to 130 as expressed clinically, *i. e.*, a marked so-called "hyperacidity." Yet this is pure gastric juice from normal persons, and this gastric juice in large quantities in the stomachs of normal persons produces no noticeable symptoms.

Whence the prevalent view that the acidity of human gastric juice is around .2 of one per cent. or from 40 to 60 as expressed clinically? This acidity, usually ascribed to pure gastric juice, is the acidity of the contents of the empty stomach, which represents the continuous secretion, plus swallowed saliva, plus occasional admixture of alkaline intestinal contents. Physiological hyperacidity does not exist. *There is no type of deviation from the normal in which the gastric glands are capable of secreting a juice of greater than normal acidity.* There may be hypersecretion but no physiological hyperacidity. The deviations in gastric juice acidity from the normal are always in the direction of hypoacidity down to complete anacidity or achylia. It is now well recognized that in so-called "hyperacidity" cases we may have gastric contents of ordinary normal acidity; we may have actual hypoacidity; or we may have an acidity approaching that of pure gastric juice, *i. e.*, from 100 to 130 as expressed clinically; yet these three conditions may be associated with the same subjective and the same gastric symptoms. Evidently the so-called "hyperacidity" is itself a symptom and not a cause of any of the prominent elements of the syndrome.

(b) *Ammonia*.—Singularly enough, the pure gastric juice con-

tains a considerable amount of ammonia, in the average of about two and one-half to three and one-half mgr. per 100 c.c. of juice, a quantity about ten times as high as in the blood. In some individuals gastric juice ammonia runs very much higher constantly or 10-12 mgr. per 100 c.c. of juice. The significance of this gastric juice ammonia is still obscure. High protein diet increases it. It is also increased by increasing the ammonia of the blood. It is increased in many cases of gastric ulcers and cancers, but we cannot as yet predict its value as a diagnostic sign in any of these conditions. The ammonia, of course, is present as ammonium chloride because of the free hydrochloric acid in the juice.

(c) *Solids*.—In general, human gastric juice is of very constant composition, both as regards acidity, salts and concentration of other solids. The total solids vary from 0.55 to 0.65 per cent., of which about 0.4 of one per cent. is organic material, probably mostly pepsin. The osmotic concentration of gastric juice is about equal to that of the blood.

(d) *Pepsin*.—The pepsin concentration is of special importance. The U. S. Pharmacopoea defines "100 per cent. pepsin as a preparation capable of digesting 3,000 times its own weight of finely divided egg white (coagulated) in three hours." When this test is applied to pure gastric juice from normal persons it is found that one to one and one-half c.c. of the juice digests 10 grams of coagulated and finely divided egg white in three hours practically as completely as is done by three and one-third grams of 100 per cent. pepsin. We have shown, partly from direct experiments on man and dogs, and partly through indirect estimation, that the adult normal person, if hungry, secretes on the average of 600 to 700 c.c. of gastric juice on an average palatable dinner, or a total of about 1,500 c.c. of gastric juice in twenty-four hours. That is to say, there is a secretion of 240 to 250 mgrs. of pepsin per dinner capable, under proper conditions, of digesting from 630 to 750 grams of protein (coagulated and finely divided egg albumen) in three hours. The total pepsin secretion in twenty-four hours is capable of digesting one and one-half kilo coagulated egg white in three hours. It is clear that the normal human stomach secretes pepsin far in excess of the actual needs of digestion. This excess of pepsin in normal gastric juice

probably explains the clinical findings of great reduction in pepsin contents without any evidence of impaired gastric digestion. It probably also explains the practical uselessness of commercial pepsin as a therapeutic measure in gastric indigestion.

We have reasons to believe that the disorders in gastric digestion, motility or sensations associated both with achylia and so-called hypersecretion and hyperacidity, are not caused to any appreciable degree by the absence or diminution of the juice, or the excessive quantity or high acidity of the juice, but they point back to a deeper seated origin.

THE BACTERIA IN HUMAN GASTRIC JUICE.

In the gastric fistula man, where no saliva can enter the stomach because of the complete esophageal stenosis, we may wash out the stomach repeatedly with sterile water and collect the appetite gastric juice under sterile conditions, yet this pure gastric juice is never free from bacteria even when it is of full acid strength (0.5 per cent. HCL). There is no question but that the development of some bacteria is greatly retarded, and that some are actually killed, by the acid gastric juice; but the practical importance of this antiseptic action has been overestimated. Water and liquid, as well as solid foods, frequently enter the intestine without development of significant gastric acidity. And so far as we know hereditary achylia cases suffer no disorder that has been definitely traced to deficient bacteriolysis in the stomach.

"An army travels on its stomach" is a trite saying and only half true. An army well fed, but with no spirit to conquer, will not travel far in the direction of the enemy. In the last analysis, an army conquers by the quality of its morale, not by the excellence of its beef stew. We have plenty of instances where underfed and ragged troops, animated with the justice of their cause and with steadfast devotion to an ideal, have bested well fed and well groomed armies with no national or ideal interest in the issue.

But, while the stomach of the army is not the whole thing, it is a factor of great importance in making a real soldier. In times of peace and plenty we may diet with fair success according to common experience and simple empiricism, but facing, as we do now, supreme stress, coupled with scarcity of sustenance,

we must have at our immediate service all the important data touching alimentation and nutrition, particularly in view of the ever increasing refinement and degradation of our natural food-stuffs. This is the excuse for bringing before you these aspects of the physiology of alimentation which have occupied our attention during the last five years.

TRANSITIONAL LEUCOCYTOSIS AND ITS DIAGNOSTIC VALUE IN CHRONIC APPENDICITIS.

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NEW YORK.

It is needless to emphasize the fact that there are occasions when it is difficult to correctly diagnose chronic appendicitis. In some instances after the most earnest attempts to establish this diagnosis, the clinician seeks an exploratory laparotomy, and, occasionally, the surgeon, too, is unable, even on inspection, to decide whether the removed appendix is diseased or not, and consequently consults the pathologist.

Clinically the tenderness elicited both by pressure on McBurney and Morris' points and by the methods of Meltzer and Bastedo, is well known to the internist and is frequently of great aid in the diagnosis of acute appendicitis. This is not, however, of such value in chronic cases, and, furthermore, does not always indicate true appendicular disease. It may mean often, for instance, periappendicular adhesions. Moreover, Roentgen examination of the appendicular region is not sufficiently conclusive. One must agree with Carman and Case that although stagnation of the contrast meal in the appendix, or a visualized appendix in roentgenograms, does not necessarily mean a pathological appendix, yet an appendix may be markedly diseased without being visible in the roentgenograms. Other Roentgen signs of appendicitis, such as pressure points in the ileocecal region, spastic colon, intestinal stasis and even shadows of concretions in the appendix do not always indicate appendicitis.

A hyperleucocytosis or a polynuclear leucocytosis, or both, when present, are valuable diagnostic aids in acute appendicitis, as their presence frequently points to a really more or less acutely diseased appendix. However, such findings are too often absent in chronic appendicitis. Occasionally, however, a leucopenia may be found both in acute and chronic appendicitis.

The routine examination of the blood of the so-called dyspeptics, many of whom were actually suffering from chronic appendicitis, suggests that in the differential count there is an important aid in the diagnosis of chronic appendicitis provided an enumeration of the large mononuclears and the transitional leucocytes is made. There is a definite increase in the number of these cells in the blood of those suffering from chronic appendicitis, although this increase may be slight at times. It may be justly designated as a transitional leucocytosis. Although not an invariable finding, it has its definite value. It is found in chronic appendicitis more frequently than a hyperleucocytosis or a polynuclear leucocytosis and more often than the positive Roentgen signs. It may be actually observed and clinically demonstrated that a transitional leucocytosis points to an appendicular disease. A transitional leucocytosis is absent in peptic ulcer, cholecystitis, renal colic, etc. (See Tables IV. and V.). If present in these or in other abdominal conditions, there is a chronic appendicitis complicating the existing condition.

The transitional leucocytes differ from the large mononuclears in that the nucleus of the transitional forms assume a horse-shoe shape. Hematologists disagree as to the proper grouping of the transitionals when enumerating them in the differential count. Wood, for instance, believes that the transitional leucocytes should be classed with the neutrophiles, and should not be considered as a separate group. Naegeli, on the other hand, states that the transitionals and the large mononuclears belong to the same group, and should be thus enumerated. Naegeli's claim is substantiated by the fact that there is no difference either in the protoplasm of the transitionals and the large mononuclears or in the structure of the nuclei, save for their form and curving. Pappenheim, however, was the first to demonstrate that a transition from mononuclears to neutrophiles never occurs and, therefore, mononuclears must be considered as a special ripe form. The percentage of transitionals in normal blood according to Wood varies from 2 to 4 per cent., while the percentage of large mononuclears is 1 per cent. These percentages seem to be quite constant. The total number of transitionals and large mononuclears in 100 white blood cells may, therefore, be considered as 5. The absolute number of large mononuclears and transitionals in a normal white cell count of 10,000 per c. cm.

will be 400 transitionals and 100 large mononuclears. In other words, the transitional formula, sometimes called relative transitional formula, will be 5, and the absolute transitional formula 500. The terms relative formula and absolute formula will be used to indicate the relative and absolute increase of leucocytes belonging to the transitional group. In chronic appendicitis the absolute formula, although frequently high, does not necessarily run, for obvious reasons, parallel with the relative formula. Rieux found a marked increase in large mononuclears (absolute numbers in the blood from 1,300 to 1,900) in severe perityphlitis. But he, like Naegeli, grouped the transitional forms with the large mononuclears. I was not aware of Rieux's findings when in a previous communication I first emphasized the importance of an increase of large mononuclears and transitionals as a diagnostic aid in chronic appendicitis. The blood in all instances was collected for examination before breakfast, or six hours after a meal, in order to exclude a digestive leucocytosis, and not less than 200 cells were enumerated. All were stained by the Jenner method. A white cell count above 10,000 per c. cm. is considered a hyperleucocytosis and in the differential anything above 75 per cent. a polynuclear leucocytosis. A count below 5,000 white cells to the c. cm. indicates a leucopenia. In nearly all cases the blood of adults was examined.*

An adequate explanation cannot be given at present for the extreme frequency of a transitional leucocytosis in chronic appendicitis. According to all hemotologists large mononuclears are produced in the bone marrow and as transition forms belong to same group, they, too, possibly, have the same origin. It may be suggested, however, that the transitional leucocytosis in chronic appendicitis is due to a constitutional disturbance which may possibly also predispose to appendicitis. This idea is strengthened by the facts that a hyperleucocytosis when present before operation, disappears after removal of the appendix and a transitional leucocytosis often does not disappear for months and even years after the appendectomy. This persistence of a transitional leucocytosis recalls that the erythrocytosis described as often occurring in chronic non-bleeding duodenal ulcer, which also

*To shorten tables, the complete counts have purposely been omitted as small lymphocytes, large lymphocytes, eosinophiles and basophiles have no direct bearing.

TABLE I. APPENDICITIS. WHITE BLOOD AND OPERATIVE FINDINGS.

Case Number.	White Blood Corpuscles.	Polynuclears %.	Large Mononu- clears %.	Transitional Leu- cocytes %.	Relative Transi- tional Formula.	Absolute Transi- tional Formula.	Operative Findings.
1	9,200	35.5	2	5	7	644	Constricted and adhesions.
2	16,800	75.5	6	9	15	2,520	Chronic and adhesions.
3	4,600	67	3.5	3.5	7	322	Constricted and adhesions.
4	7,800	65	5.5	5.5	11	858	Gangrenous.
5	24,000	77.5	6	4	10	2,400	Fibrinous.
6	8,400	55	2.5	11	13.5	1,134	Chronic
7	6,000	57	1	6	7	420	Chronic.
8	8,000	72	1	6	7	560	Chronic.
9	8,000	62	3	7	10	800	Chronic and adhesions.
10	11,300	68	0.5	2.5	3	339	Chronic.
11	11,000	42	6	0	6	660	Chronic and adhesions.
12	12,600	79	7	3	10	1,260	Retrocecal and adhesions.

TABLE II. APPENDICITIS: WHITE CELL COUNT: ROENTGEN AND OPERATIVE FINDINGS.

Case Number.	White Blood Corpuscles	Polynuclears %.	Large Mononuclears %.	Transitional Leucocytes %.	Relative Transitional Formula.	Absolute Transitional Formula.	Roentgen Findings.	Operative Findings.
1	11,800	54	2	4.5	6.5	767	Stagnation of barium in terminal ileum.....	Chronic and adhesions.
2	10,000	65	6	5.5	11.5	1,150	No signs.....	Fibrous.
3	7,200	59	2	5	7	504	Spasticity of colon.....	Constricted.
4	14,800	75	3	4	7	1,036	Tender point in ileocecal region.....	Chronic and adhesions.
5	7,400	68	1	1.5	13.5	999	Intestinal stasis-visible appendix.....	Long appendix. Mucosa ulcerated.
6	9,600	49	1	7	8	768	Visible appendix.....	Chronic and numerous adhesions.
7	11,200	63	4	2	6	672	No signs.....	Chronicobliterativeandadhesions.
8	6,600	72.5	2	5	7	462	Spasticity of colon.....	Chronic.
9	4,200	51	6	13.5	19.5	819	No signs.....	Perforated.
10	6,400	57	0	5	7.5	512	No signs.....	Fibrous.
11	8,600	58	0	5	0	43	Tenderpointin ileocecal region visible appendix.....	Kinked and adhesions.
12	6,400	42	3	9	12	768	No signs.....	Chronic.
13	5,400	57	4	4	8	432	No signs.....	Normal with adhesions.
14	11,600	62.5	4	3.5	9.5	1,102	Visible appendix.....	Constricted.
15	6,800	45	6	9	15	1,020	Visible appendix.....	Chronic.
16	10,400	42.5	6	1.5	4.5	572	Visible appendix.....	Kinked and adhesions.
17	7,200	37	0.5	0	0.5	36	Adhesions about appendix.....	Retrocecal and adhesions.
18	6,000	61	1	1.5	1.5	180	Intestinal stasis.....	Chronic.
19	9,900	62	1	6	7	693	No signs.....	Constricted and adhesions.
20	5,000	51	3	5	8.5	425	Visible appendix.....	Chronic and concretions.

TABLE III. WHITE CELL COUNT IN APPENDICITIS ASSOCIATED WITH OTHER CONDITIONS:
ROENTGEN AND OPERATIVE FINDINGS.

Case Number.	White Blood Corpuscles.	Polynuclears %.	Large Mono- nuclears %.	Transitional Leu- cocytes %.	Relative Transi- tional Formula.	Absolute Transi- tional Formula.	Roentgen Findings.	Operative Findings.
1	6,200	62	1.5	4.5	6	372	Adhesions about appendix.	Chronic and adhesions. Strawberry gallbladder and peri-cholecystic adhesions.
2	4,400	66	0	2	2	88	Tender point in ileocecal region. Visible appendix.	Buried in adhesions. Chronic cholecystitis with adhesions.
3	5,200	60	4	7	11	572	No signs.	Kinked with concretions—numerous adhesions around. Callous ulcer at pylorus posteriorly. Thick gallbladder with several stones.
4	4,800	41	4	6	10	480	Tender point in ileocecal region.	Chronic indurated ulcer at lesser curvature near pylorus. Adhesions between gallbladder and pylorus.
5	6,900	70	4.5	3.5	8	552	No signs.	Mucosa inflamed and ulcerated. Broad adhesion band. Callous ulcer at first portion of duodenum anteriorly.
6	7,100	61	3	5	8	568	No signs.	Constricted with adhesions. Callous ulcer at first portion of duodenum anteriorly.
7	8,000	50	7	5	12	960	No signs.	Adherent with inflamed mucosa. Callous ulcer at first portion of duodenum with small perforation.
8	3,800	59	5	1	6	228	No signs.	Constricted and adhesions. Callous ulcer at first portion of duodenum anteriorly.
9	7,400	50	1	6	7	518	Tender point in ileocecal region.	Chronic. Broad band around colon causing intestinal obstruction. Diverticulum in the colon.
10	5,400	56	1	1	2	108	No signs.	Chronic. Ulcer between second and third portion of duodenum.
11	9,400	52	1.5	6.5	8	752	No signs.	Kinked, thickened and bound by adhesions. Scar at extreme portion of pylorus.
12	4,600	66	0	2.5	2.5	115	No signs.	Long appendix. No inflammation. Adhesions at mesenterium and between gallbladder and pylorus.
13	7,400	44	4	4	8	592	Tender point in ileocecal region.	Chronic and adhesions. Perforated duodenal ulcer.

TABLE IV. WHITE CELL COUNT IN CHRONIC PEPTIC ULCER AND OPERATIVE FINDINGS.

Case Number.	White Blood Corpuscles.	Polynuclears %.	Large Mononuclears %.	Transitional Leucocytes %.	Relative Transitional Formula.	Absolute Transitional Formula.	Operative Findings.
1	6,200	68.5	0	1	1	62	Callous ulcer at lesser curvature.
2	7,600	68	0	1	1	75	Indurated ulcer with adhesions.
3	6,200	71.5	0	0.5	0.5	31	Callous ulcer at pylorus. Appendix appeared normal.
4	9,400	63	0	1	1	94	Callous ulcer at lesser curvature. Appendix was normal.
5	12,000	76	1	0	1	120	Perforated ulcer at posterior wall of stomach.
6	6,800	63.5	0.5	1	1.5	102	Callous ulcer at first portion of duodenum. Removed appendix normal.
7	9,600	71.5	0.5	2	2.5	240	Soft ulcer at first portion of duodenum. Appendix normal.
8	5,800	68	1	1	2	116	Pyloric obstruction due to large callous ulcer. Appendix normal.
9	7,200	60	1	0	1	72	Callous ulcer at first portion of duodenum. Appendix normal.
10	9,300	0	3	3	279	Large indurated ulcer at posterior wall of duodenum. Appendix normal.

TABLE V. WHITE CELL COUNT IN MISCELLANEOUS CONDITIONS AND OPERATIVE FINDINGS.

Case Number.	White Blood Corpuscles.	Polynuclears %.	Large Mononuclears %.	Transitional Leucocytes %.	Relative Transitional Formula.	Absolute Transitional Formula.	Operative Findings.
1	9,200	55	0	2	2	184	Peri-cholecystic adhesions.
2	16,800	84	1	0	1	168	Thick gallbladder and stones.
3	5,600	65	1	1	2	112	Hour glass stomach caused by adhesion band extending from liver to stomach.
4	11,400	74	6	5	11	1,254	Gallstones. Fistula between gallbladder and colon.
5	23,000	75.5	0	5	5	1,150	Perforated duodenal ulcer right-sided subphrenic abscess.
6	5,000	68.5	0.5	0.5	1	50	Sclerosis of pancreas.
7	11,200	59	2	0	2	224	Gallstones.
8	6,900	64	0	0	0	0	Gallstones.
9	7,600	71	1.5	1.5	3	228	Stone in left kidney.
10	6,000	68	2	0	2	120	Stone in right kidney.

TABLE VI. WHITE CELL COUNT IN PATIENTS WHOSE APPENDICES HAD BEEN PREVIOUSLY REMOVED.

Case Number.	White Blood Corpuscles.	Polynuclears %.	Large Mononuclears %.	Transitional Leucocytes %.	Relative Transitional Formula.	Present Illness.
1	12,400	63	6	0	6	Perforated duodenal ulcer.
2	8,200	54	6	1	6	Floating kidney.
3	55.5	2.5	6.5	9	Postoperative adhesions around appendix.
4	5,600	66.5	3	5	8	Caecum mobile.
5	9,200	71	2	1	3	Postoperative adhesions and intestinal stasis.
6	12,800	60.5	4	6	10	General visceroptosis.
7	10,000	40	3	3	6	Ascaris lumbricoides.
8	4,000	55	2	6	8	Bleeding gastric ulcer.
9	5,400	60	0	6	6	Postoperative peri-appendicular adhesions.
10	9,400	52	0	8	8	Asthenic gastric catarrh.
11	6,100	68	2.5	0	2.5	Spastic constipation.
12	4,200	61	0	0	0	Hepatoptosis.
13	71	0	5	5	General itching of body. Origin unknown.
14	5,400	60	0	0	0	Postoperative adhesions.

probably is due to a constitutional disturbance, likewise persists after the various surgical procedures.

The present study is based upon sixty-five cases. Fourteen of these presented themselves for examination for ailments other than appendicitis, their appendices having been previously removed at the advice of other physicians. (See Table VI.) For the operative findings I am indebted to the following surgeons: Dr. John A. Bodine, 29 cases, Dr. A. O. Wilensky, 11 cases; Dr. Leo Buerger, 10 cases; Dr. A. A. Berg, 7 cases; Dr. Charles N. Dowd, 2 cases; Dr. Charles Peck, 2 cases; Dr. M. S. Kakels, 1 case; Dr. James L. Russell, 1 case; Dr. Charles R. Hancock, 1 case, and Dr. John Deaver, 1 case. For the pathological reports of ten cases I am indebted to Dr. C. H. Bailey.

In Table I. the relative formula is found to be in the majority of cases above 5 and the absolute formula above 500. In these patients the appendicular region alone was explored at operation since the symptoms and signs did not point to any other organic

disease within the abdomen. Roentgen examinations were not made. In all but one the high relative formula proved to be of definite diagnostic value.

Of the twenty cases listed in Table II. in but one (Case 13) the appendix was found on gross and microscopical examination to be normal, even though a high relative formula was found. It is also observed that out of the nineteen patients in whom a diseased appendix was found, Roentgen signs were positive in thirteen. A hyperleucocytosis was noted in five patients, polynuclear leucocytosis in none and leucopenia in one.

ABSTRACTS OF HISTORIES OF ILLUSTRATIVE CASES.

CASE I. (Case 15, Table II.) H. G., female, aged 15 years; first seen April 6, 1918. Complained of obstinate constipation. Two weeks previously she had an attack of pain in the right lower quadrant of the abdomen. Physical examination revealed marked tenderness in the appendicular region, which was also elicited by Meltzer's method. The appendix was visible six hours after ingestion of the contrast meal and remained visible in the twenty-four and forty-eight hour plates. The history is a typical one; the physical and Roentgen signs were positive and the transitional formula high—15.

CASE II. (Case 1, Table II.) A. O., female, aged 18 years; first seen December 28, 1915, complained for the last six years of pain in the pit of the stomach and of obstinate constipation. The pain was always immediately relieved by eating. She vomited on several occasions. On physical examination there was slight tenderness in the epigastric region. No tenderness was elicited by palpation in the appendicular region or by Meltzer's method, and digital rectal examination was negative. There was a slight microscopical stagnation of food on fasting stomach and a marked hyperchlorhydria after an Ewald's test breakfast. The stools were positive for occult blood on three different occasions. Fluoroscopic examination and radiographic findings showed a subtonic type of stomach; no tender pressure points at curvatures of the stomach or at the pyloric ring; no niche and the duodenal cap regular and well filled. There was a large gastric residue nine and one-half hours after ingestion of the barium meal and the terminal ileum was found to be slightly filled twenty hours later. The appendix was not visible. A diagnosis of gastric ulcer was made. The patient was put on a Lenhart diet for two weeks. She improved and remained well under Ulcus therapy until April 15, 1916, when all the symptoms returned. Operation May 25, 1916, revealed chronic inflammation of the appendix with peri-appendicular adhesions. The gall-bladder and pancreas were normal and no ulcer found in the stomach or duodenum. It is quite evident that this case simulated gastric or duodenal ulcer. The diagnosis of appendicitis

was not made prior to the operation because the value of the transitional leucocytosis was then underestimated.

CASE III. (Case 1, Table III.) A. H., male, aged 31 years, first seen September 18, 1917. For the past eleven years he suffered from indigestion and attacks of pain in the pit of the stomach which radiated to the lower right quadrant of the abdomen; pain had no relation to food and was more intense on laughing, sneezing or coughing. He vomited with blood at the onset of his illness. He was treated at Wegele's Sanitorium in 1917 for gastric ulcer and the same diagnosis was made by Professor Adolph Schmidt in Frankfurt. On examination he showed tenderness in the continuation of the right axillary, and scapulary lines at the level of the gall-bladder and marked tenderness in the appendicular region by Meltzer's and Bastedo's methods. There was no stagnation of food and the acidity of the stomach contents was normal. Occult blood tests of the stools were repeatedly negative. Urine analysis, Wassermann reaction, fluoroscopic and radiographic examination of the stomach were negative. There was a stasis in the large intestine seventy-two hours after ingestion of the barium meal, and after a barium enema the transverse colon appeared to be markedly ptotic, sharply kinked at the proximal portion and adherent to the cecum. A diagnosis of pericholecystic adhesions and chronic appendicitis was made. At operation the appendix was found buried in adhesions. A strawberry gall-bladder with adhesions was also removed. There was no ulcer of the stomach or duodenum and the pancreas appeared to be normal. Transitional leucocytosis, tenderness elicited by Meltzer's and Bastedo's method, Roentgen signs and the tender pressure points in the right axillary scapulary line at the level of the gall-bladder, all added to make the diagnosis of appendicitis and pericholecystic adhesions.

CASE IV. (Case 4, Table III.) W. M., male, aged 43 years, first seen October 31, 1917, had suffered for a number of years with vomiting and sharp pain in the pit of the stomach immediately after meals. Examination revealed tenderness in the epigastric region. The laboratory and Roentgen findings pointed to an ulcer of the stomach. There were no symptoms or Roentgen signs or other physical findings to lead one to suspect an associated appendicitis. At operation, a large indurated ulcer was discovered at the lesser curvature near the pylorus. There were adhesions between the gall-bladder and the duodenum. The appendix was removed and found to be normal on gross examination, but microscopically scattered through the mucosa were many phagocytic cells filled with hemosiderosis pigment. The epithelium was normal, but there was some fibrous thickening of the submucous coat and occasional fibrous scarring of the muscular coats. These changes suggested a mild chronic appendicitis. The high transitional formula was the only aid for the diagnosis of appendicitis.

CASE V. (Case 2, Table III.) K. G., male, aged 29 years, first seen April 2, 1918, gave a history of indefinite abdominal pain appearing at intervals of several months. He had his first attack five years

ago. Since then he was observed by several internists and a complete Roentgen examination of the gastro-intestinal tract had been made. A definite diagnosis was not reached. However, based upon a transitional formula of 8, a positive diagnosis of appendicitis was made in spite of physical and Roentgen signs. At operation, April 20, 1918, a kinked and thickened appendix with adhesions was found and a scar resulting from a healed ulcer at the extreme portion of the duodenum was discovered. Microscopically some fibrous thickening of the muscular coat with slight fatty replacement was observed.

CASE VI. (Case 2, Table III.) F. B., female, aged 30 years; first seen September 29, 1917, complained of indigestion for many years, her condition becoming worse during the past year. Her chief complaint was a sensation of pressure or load in the epigastrium. She was extremely nervous, suffered a great deal from obstinate constipation, alternating with diarrhea, and in the course of one year she had lost 40 pounds in weight. Various diagnoses were made, such as nervous dyspepsia, autointoxication, and even acidosis. She appeared emaciated, her weight being 88 pounds. She showed a marked dermatographia and had extreme tenderness in the continuation of the axillary line at the level of the gall-bladder. Tenderness was also elicited in the appendicular region on palpation by Meltzer's method and on rectal examination. There was no stagnation of food, but the free hydrochloric acid was strongly positive. The stools and urine analysis were negative except for slight traces of sugar on several occasions. Indican was always in excess. Fluoroscopic and radiographic examinations showed marked ptosis of the stomach and of the transverse colon. The appendix was visible in 24, 28 and 72-hour plates. A diagnosis of cholecystitis was made. Appendicitis was not entertained because of the low transitional formula, 2, although there was intestinal stasis and a visible appendix. At operation, October 19, 1917, adhesions between gall-bladder, duodenum and pylorus were found. The gall-bladder was atrophic. Cholecystectomy was performed. Adhesions were noticed around the appendix, which was removed. On microscopical examination, however, the appendix was normal. This case illustrates that physical and Roentgen signs may be present without an actually diseased appendix, but the low transitional formula helped, indeed, to rule out appendicitis.

SUMMARY.

1. Transitional leucocytosis, or an increase in large mononuclears and in transitional leucocytes, or an increase in either of them, was found in the blood of 87 per cent. of patients in whom evidence of chronic appendicitis was obtained.
2. There was no transitional leucocytosis in the blood of patients in whom evidence of chronic peptic ulcer was obtained,

or in the blood of those in whom cholecystitis, renal stones, or other organic abdominal conditions were found at operation.

3. A transitional leucocytosis was found in patients in whom appendicitis was present with other organic abdominal conditions.

4. A hyperleucocytosis and a polynuclear leucocytosis are not as frequently found in chronic appendicitis as a transitional leucocytosis.

5. A transitional leucocytosis as a diagnostic aid is superior to such Roentgen signs which are supposed directly or indirectly to point to a diseased appendix.

6. Transitional leucocytosis often persists in the blood after an appendectomy is performed.

1000 Park Avenue.

THE MENTAL FACTOR IN THE CHRONIC INTESTINAL
INVALID.

BY JOHN BRYANT, M. D.,

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At the last annual meeting of this Association, Dr. Jacob Kaufmann gave a most instructive short paper on the "Psychic Element as an Important Factor in the Development and Treatment of Peptic Ulcer." He stated that "as a rule we are dealing here with high-strung, excitable individuals who are prone to indulge in over-activity." He referred to the fact that "the intimate connection between the mental status and digestive activity and its disturbances, long known to the laity as well as to medical observers," had been experimentally demonstrated by many able workers among whom he mentioned Pawlow and Cannon. Later in his paper, the following paragraph appeared:

"Whatever other medical or surgical treatment may be indicated, according to the condition which the case presents, the final result, especially the prevention of recurrences, depends to a high degree on the possibility of eliminating further periods of display of harmful psychic influences. With this object in view, we must enlighten the patient on his shortcomings, we must advise him to avoid emotional excesses and to redress faulty habits of mental activity. This may involve a thoroughly revised mode of living and eventually necessitate a change of occupation."

These remarks of Dr. Kaufmann's were applied to a consideration of treatment of peptic ulcer. They apply no less to many other classes of cases, and especially to the chronic intestinal invalid with whom I have been concerned in recent years.

The object of the present paper is to give two case histories which demonstrate the applicability of Dr. Kaufmann's thesis to the chronic intestinal invalid. The term CHRONIC INTESTINAL INVALID is applied to the individual who is usually in rather poor health from an intestinal point of view. Many of these individuals do not even associate their poor health with intestinal abnormalities, due to the fact that they have very little gastric distress; also, because they have a daily bowel movement,

the idea that this movement may be one or more days late in arriving never having been even considered. There is often on the other hand, a clear history of intestinal trouble dating back to childhood, as in Case 1 here reported. Also, as in Case 1, which is the third generation to suffer from intestinal trouble, heredity frequently seems evident.

The degree of poor health is very variable. Although many of these chronic intestinal invalids may get along well for months at a time under favorable conditions, excessive fatigue, either mental or physical, almost invariably precipitates a train of symptoms among which abdominal pain, and intermittent diarrhea or constipation predominate. Accompanying this more purely gastro-intestinal display of symptoms, is a condition of general bodily exhaustion varying in degree according to the length of time for which the particular patient has been under strain. From the point of view of physical type, the chronic intestinal invalid is usually "high-strung and excitable"; this mentality is usually heralded by a long, thin, often emaciated carnivorous type of body. The skin is usually pallid or sallow and may be so discolored as to suggest Addison's disease. As the long, thin body would lead one to expect, this type of individual often presents a text-book picture of drop-heart and all the other classic features of visceroptosis as usually described. The posture is bad. There may be a drawn, anxious facies. The circulation is poor, the blood pressure being often low. The patient often suffers from cold hands and feet, and frequently accompanying the low blood pressure and the cold hands and feet, there is a disagreeable mottled purplish color of the hands and feet which may extend almost to the elbows and knees. Perspiration, not infrequently disagreeable in odor, is often so excessive as to suggest hyperthyroidism; the palms, and the soles of the hands and feet are in these persons often not only cold but clammy. As a whole, although the brain may be hyperactive, the body usually has physical inefficiency written all over it, even in the absence of demonstrable organic lesions.

The two cases about to be described were seen recently while in the Army. One is a nurse, and one is the wife of a higher Army officer. It is only accidental that both of these recent cases are of the same sex. There are many men who fall into this category of the chronic intestinal invalid, as anyone who has had gastro-intestinal practice can testify.

CASE I. Mrs. G. P. A., white, officer's wife, age 56, entered Walter Reed General Hospital, December 6, 1918.

Family History.—Father died of bowel trouble at the age of 33, after an illness of three or four years during which time he was unable to work. The paternal grandfather is said to have had the same sort of trouble, and to have died of it suddenly. Mother's family all well.

Past History.—First began having trouble when six years old. Sharp attacks of pain and diarrhea about two or three times yearly, lasting from two to three days. In intervals, fairly healthy. At 16-17 years of age the attacks became more frequent and "colds would settle in the bowels." Menstrual history began at the age of 15, very stormy, always great pain, in bed for twenty-four hours, quantity slight; gradual improvement after thirty years of age. Patient was married at 28 but had no children; this was attributed to a run-away accident and the severe nervous state which followed. Underwent several operations for sterility but without effect. At 32 was operated upon in New York for "ulcers of the rectum" and the ulcers were burned. This relieved for a time the severe and more or less constant rectal pain. This operation occurred while the patient was in hospital for displacement of the uterus.

The abdominal pain, either general or in the right lower quadrant, gradually became worse. Five years ago, patient had an interval appendix removed. Has been more free of localized pain since, but there has been no improvement in the general abdominal pain associated with dysenteric spells. These have come six or eight times a year, lasting several days to two weeks, at which times patient always gets very weak and has bearing down pains. The rectal pain is occasionally severe enough to require morphine. Patient sleeps poorly, often remaining awake after two or three o'clock in the morning. Appetite is good but is afraid to eat on account of bowel condition. She has avoided coarse or rough foods, but has otherwise used a general diet with care. For the past six months since conditions have been worse, has lived on eggs, toast, rice and broth, as other foods seemed to aggravate her trouble. The bowels have moved at least about four times daily, the movements being ropy and frequently containing mucus. When she is worse the movements are watery with much mucus. Patient lived for fifteen years in the Philippines, returning three years ago. While in the Philippines, about seven years ago, she was known to have been suffering from amoebic dysentery. This is proven to have been cured.

Present Illness.—At the time of admission to the hospital, December 5, 1918, patient was having from eight to fifteen movements daily associated with gas, watery material and mucus; also complaining of more or less constant pain in rectum, especially at night. Patient entered hospital determined that she must undergo a serious intestinal operation of some sort for relief of her present condition, her feeling being that anything would be preferable to her then constant state of misery and inability to carry on her normal daily life at home.

Physical Examination.—In general, negative, except that patient is very thin. Abdomen generally tympanitic with general soreness but no local tenderness. Skin dry, discolored.

Patient first seen by writer on December 21, 1918. Abstract of notes follows.

December 30. Has been on a special diet, omitting all eggs, meat and fish, for past five days. In general, improved. Less bowel frequency, less pain and rectal tenesmus, also less gas. Yesterday there was a slight food upset, but patient is better today.

January 4. Steady improvement. Now has no abdominal discomfort and little gas. Sleeps much better. Appetite satisfactory. Bowels have moved only once daily for the past forty-eight hours instead of nine or ten times daily. Feels stronger and much more cheerful. Given exercises to enlarge lower thorax, also general developmental exercises. Given massage daily.

January 7. Feels well, good appetite, sleeps well, bowels regular once daily, normal in quantity and appearance, walking about, desires to go home.

January 24, 1919. Discharged to home, improved.

This patient when first seen stated emphatically that it was impossible for her to eat ordinary vegetables. Within two weeks of the time of alteration of diet, she was eating everything allowed on her prescribed diet, which eliminated, practically speaking, only eggs, meat and fish.

At the time of my first conversation with the patient, she understood me to say that because I did not wish her operated upon, her case was hopeless. It took several days to disabuse her of this idea, so firmly fixed had it been in her mind that she must be operated upon in order to obtain any relief from her sad condition.

Two weeks after being first seen, patient said the change produced in two weeks "is so great it seems as though a miracle had been performed." She expresses profound and almost embarrassing gratitude.

She responded in general very readily to mental or nervous treatment. This is shown by an incident which happened on the day when it was first attempted to give her some exercises which would tend to enlarge her lower thorax. After three or four long breaths she suddenly complained of terrific pain over the whole lower thorax, especially in the epigastrium. This was associated with a semihysterical spasticity of the fingers. The mechanism of this finger "tightness" from which she had suffered

for years was explained to her. It disappeared almost before the explanation was completed, and it never returned. The epigastric pain, following exercises, persisted violently for twenty-four hours. Exercise was resumed after forty-eight hours without further incident, and at the time of leaving the hospital, patient's posture had already become much improved.

About six months after returning home, this patient informed me that for the first time for years she had known what it was to be able for weeks at a time to run up and down stairs at will, practically without fatigue, and free from her long-standing intestinal ailment.

Several months after her leaving the hospital, she suffered from an acute food upset. She was able, however, to overcome this herself by a strict application of the dietary system which she had been taught, and at last accounts she was back again at her daily work feeling "better than for many years."

Summary Case I.—¹ An emaciated elderly woman who had suffered from recurrent intestinal attacks since childhood, entered hospital seeking operation in a spirit of desperation at her state of chronic invalidism as evidenced by extreme rectal tenesmus, abdominal pain, and more or less chronic dysentery to the extent of eight to fifteen dejections daily. She sought relief from a condition summed up by her as "hopeless invalidism" in which she was a burden to herself and to her husband.

2. Treatment: Diet, exercise, advice, massage.

3. Results: a. After fourteen days, bowels moving once daily, practically no gas, no abdominal discomfort, no rectal tenesmus, mental state more placid.

b. After one month, eager to go home, discharged from hospital. Able to do her own housework and carry on the daily routine of her life without undue fatigue.

c. After six months, no relapse.

CASE II. Miss E. V. N., nurse, white, age 24, on duty at the Walter Reed General Hospital, was admitted as a patient on April 24, 1918.

Family History.—Negative except that the patient was one of a large family and for some years had felt the necessity of earning her own living.

Past History.—The only childhood complaint remembered was diphtheria. The patient was operated upon for appendicitis when nineteen years of age, at which time a dermoid cyst of the right ovary was also removed. The following year, patient for several weeks suffered from severe headache, nausea and vomiting; she had to go to bed on account of severe darting pains in the hepatic region, and then remained off duty for ten weeks on account of constant dull pain follow-

ing the acute pain just mentioned. This radiated backward to the side and up toward the lower end of right scapula. Two years ago patient was operated upon for correction of abnormal nasal septum and turbinate bones. In the same year, while nursing a case of delirium tremens, she was kicked in the back and at this time a kidney is said to have been torn loose. The patient wore a binder for one year and has not been troubled by kidney pain since.

Present Illness.—In January and February of the present year, patient was in the Walter Reed Hospital suffering from meningitis contracted while on duty. She recovered well, and soon went back to duty. She felt well enough until April 10th, when she became nauseated and vomited. Her temperature varied between 100-102°. There was at this time severe cutting pain in the hepatic region. Patient tried to continue on duty. The pain radiated to the side and back as in the attack she had had in 1914, in fact, the symptoms are said to have been very similar to those in the previous attack.

Patient again came on sick report on April 21. At this time the temperature was recorded at 100, pulse 95, respiration 24. The chief complaint was of dull pain in the hepatic region radiating to the side and back, increasing on motion, especially on turning in bed. Pain at this time was pulling or tearing when sitting or standing very long. Patient stated upon being admitted that she could not sleep night or day on account of itching all over her body, although there was no sign of skin trouble. There was habitual constipation and the stools were at times "pasty white."

Physical Examination.—Patient rather short in stature, well developed, somewhat obese. A very pallid color of the entire skin. Liver slightly enlarged and tender to palpation and percussion. Local tenderness over the gall-bladder. Examination otherwise negative.

One month after admission, there had been no improvement in general condition. Patient came under the personal care of the writer on May 24, 1919. Abstract of notes follows:

May 28. Much less gas and pain; no vomiting; sleeps better.

June 9. For three days has had a right mastitis without temperature.

June 15. Is again doing well; marked improvement in developmental exercises.

June 30. General condition better; local pain persists.

July 12. Increase of local pain and increase of mucus in stools, otherwise improved.

July 18. Has had severe menstrual pain, and active pain over gall-bladder.

July 21. Much improved in every way.

July 28. Slight return of pain in gall-bladder region associated with distension of cecum by gas.

July 31. Up and about. Feels much stronger, but frequent loose movements for past forty-eight hours.

August 31. Very slow gradual improvement in bowel condition.

Now has no complaint of pain aside from menstrual distress. Oil enema (four ounces being retained at night) being used with apparent good effect. Improvement in general condition and strength slow but continuous. An occasional unexplained rise in temperature to 99-100 lasting one or two days.

September 7. For two weeks has had persistent daily headache, unilateral from eye to occiput on right side; unexplained. No abdominal distress, bowels regular, movements somewhat light colored. Sleeping well and getting about with constantly less effort. In general, condition satisfactory but patient far from well and can never be fit for Army duty. Would require from three to four months furlough and would then be unable to stand hard work.

September 9. Free from headache today.

September 10. After an interval, remarkably free from intestinal pain of any sort, in which patient was up and about for a week or two dressed, pain in the right lower quadrant reappeared. This was associated with irregular menstruation which persisted only one day, followed by relief of pain. Patient then continued favorably until September 14th, when the abdominal pain recurred in the old area, right upper quadrant, just outside of gall-bladder area, radiating upward and outward toward the right axilla. Temperature to 101 for three days. White count 7,000. Mucus and streaks of blood in stools.

September 17. Pain much less acute and patient quite comfortable though still sore and tender, the pain being more localized to the ascending colon and hepatic flexure region. At noon the pain suddenly became violent, same in character as two or three months ago, requiring morphine gr. $\frac{1}{2}$ for relief.

September 22. Patient yesterday afternoon had a white count of 17,000 and temperature of 101.6, but today temperature is down to 99. Patient still very comfortable. Morphine gr. $\frac{1}{4}$ twice in the night required for pain and lack of sleep. Vomited green liquid several times and has had several spells of hiccough.

September 25. Pain slowly subsiding. Temperature has not been over 100. Very exhausted, marked herpes of lips.

October 1. Much improved but pain persists.

October 5. To have an exploratory laparotomy when influenza epidemic is passed. Patient unable to do any concentrated work, as making gauze masks, without its being followed by an undue nervous exhaustion, tremor and increase of local abdominal pain. This pain follows the course of the ascending and transverse colon.

October 9. Condition unchanged. Slight exertion provokes pain. Fairly comfortable on light diet, flat in bed.

October 15. Has had three days of discomfort associated with nausea. Sharp intestinal pain in gall-bladder region associated with mucus in stools. Today, fairly comfortable.

October 20. Greatly improved.

October 28. Out of bed for short periods the past three days, the

first time in over one month. Much brighter and more cheerful than for a long time. Today no great tenderness except in a small spot near gall-bladder area.

November 1. Steady improvement.

November 8. Better than for weeks past. Almost no local tenderness.

November 10. Transferred for exploratory laparotomy; question of intestinal adhesions, with possible involvement of gall-bladder.

November 12. Operation; Major Hill. A band of adhesions found running from the lower ascending colon to the middle third of the transverse colon, involving an hepatic loop of the upper ascending colon. No other abnormality noted. Adhesive bands cut, restoring contour of colon approximately to normal. (The operation was short and satisfactory, the patient being returned to bed with practically no post-operative sequellae.)

December 10. For four days patient has been up and about after an uncomplicated convalescence.

December 15. Discharged to duty.

These notes have been quoted extensively to show the erratic progress of the case during the approximately six months the patient was under my observation until the time of her operation. It may be said that six months was a long while to wait for operation in a case of this sort. The answer is that no one would take the responsibility for making a diagnosis. For example, an unsuccessful attempt had been made to fasten the whole series of symptoms upon neurasthenia pure and simple. This appeared to me to be nonsense, although the patient was obviously high-strung. Furthermore, in the course of the months that this patient was under control of the writer, it was possible to obtain a very considerable degree of physical development, and to largely increase the mental poise. For weeks previous to the latter half of October, the writer had felt that there was some serious mental commotion going on which would probably come to the surface if allowed adequate time. The patient had previously refused to yield any information to a psychologist.

On October 26th, after a two-hour struggle with herself, the patient unbosomed herself of a minor sexual irregularity which, although of no great consequence in itself, had seemed very terrible to her, and to which she had looked back with horror ever since early puberty. There was in addition a broken engagement of more recent occurrence. It was possible to get the patient in a perfectly comfortable frame of mind in reference to

these two events. The result was startling. The entire nervous mask fell away. In forty-eight hours the patient had become almost placid in temperament. She continued constantly and quietly cheerful throughout the remainder of her stay in hospital.

Had not the mental factor been eliminated and had not some degree of physical improvement been obtained, it is very probable that the operation might not have effected a cure. As it was, the operation was in itself rapid and successful, the patient came through with no loss of mental poise, and was soon almost upon her feet again. The word "almost" is used advisedly. Possibly on account of the writer's being at the time away on an inspection trip, the patient was allowed to be placed on active duty while awaiting her discharge from the Army. The discharge was held up, and the amount of duty was not properly regulated. As a result the patient arrived home in a somewhat exhausted condition. After a month or two of rest, she has, however, been able to take up District Nursing, and she has recently written that she is practically free from all gastro-intestinal complaints. Her only cause of present distress is fatigue from physical over-exertion, for, as she says, "Mental worries are a thing of the past."

Summary Case II.—1. A young woman of good physique, exhausted from over-work, suffering from recurrent intestinal attacks accompanied by fever, abdominal pain and mucus in the stools.

2. Treatment: Diet, exercises, advice, massage, operation.

3. Results: a. Marked increase of physical and mental poise with relief of mental distress obtained in the six months' preceding operation.

b. Operation for adhesions successful.

c. Uninterrupted recovery; discharge from hospital one month after operation.

d. After discharge from the Army, returned home; able to resume District Nursing work and to keep it up without return of gastro-intestinal distress.

DISCUSSION.

The above two cases represent two types of mental disturbance which must be taken into account in the treatment of the chronic intestinal invalid. It is presumable that success would not have been obtainable in either of these cases without adequate attention to this mental factor.

In the first case mentioned, the mental commotion present was due to a summation of all sorts of long-continued incoming stimuli

which had greatly worn down the patient's resistance, so that she was in a state of general unstable nervous equilibrium. Equilibrium was restored by a process of general re-education as well as by removal of the local intestinal irritation.

In the second case, almost the entire nervous commotion present was due to two specific causes. On removal of these two causes of irritation, the patient ceased to be nervous. It is certain that this had much to do with her uneventful recovery from operation.

SUMMARY.

1. Two case histories have been presented, illustrating two distinct types of mental commotion often encountered in the treatment of the chronic intestinal invalid. In one case, the mental commotion was due to a summation of general causes. In the other case, the mental commotion was due to two specific factors.

2. In both of the cases above presented, relief of the mental commotion proved a very important factor in the recovery of the patient.

3. Consideration of the mental factor is a prerequisite for the successful treatment of the chronic intestinal invalid.

DISCUSSIONS.

DR. F. M. POTTENGER, Monrovia, California: I wish to emphasize a point that Dr. Bryant made. There is no question but that the ordinary operation on a patient of the type just mentioned is not only useless, but often harmful. We must take into consideration the nervous and psychic conditions. These patients are poor risks. They are operated and operated, and treated for this and that; everything but the psychological condition. You can control them, if you explain to them the things that they do to upset themselves. They are upsetting things, instead of things upsetting them. We must approach them in a sympathetic way. The medical profession is wrong in its attitude towards them. You never get anywhere, unless you take them into your confidence. You must not say that their pain is not there. It is there. We cannot always explain the mechanism, but they have pain more easily than they ought to have; and if you raise the threshold of response, so that the nerves do not react so readily, you can relieve many of them. Treat them psychologically. Give them what they deserve. They need psychological treatment. The province of medicine is not alone giving medicine and operating, but treating the patient. The Christian Scientist has made us recognize that these patients are hoping human beings.

MAJOR JOHN BRYANT, Washington, D. C.: The gastro-intestinal invalid is one of a class that I call hyposthenics. They are not up to one hundred per cent. physically, and never can be. The question is, what is to be done with the large group of humanity covered by this word? About ten per cent. of them are not perfectly sane, and twenty to twenty-five per cent. of them are no good physically. In the army, well over twenty-five per cent. were refused by the draft boards, at least fifteen per cent. additional were lost in the course of training before the army went to Europe, and another twenty to thirty per cent. that went across would never be any good in the front line. Out of the total draft probably not much over thirty per cent. were eventually front line material.

The hyposthenics may be forty to sixty per cent. good, but you can never make them one hundred per cent. material. You can, however, raise them at least twenty to twenty-five per cent. above their previous level, and it is worth while to do this, as it will make all the difference between whether they can get along in their daily life or not.

Concerning the hyposthenic group as a whole, I think there are two factors of importance. The first is defective elementary education. The second is apathy or lack of ability on the part of the parents to understand their children. There are mothers who are afraid to discipline them. The average boy gets twenty years of discipline before he gets through college. At the end of that twenty years, whether he wants to or not, he has had his control apparatus formed to a considerable extent, and is then in a very different position from the patient who has been sickly, or the girl who has never had more than two or three years of real schooling.

In the way of treatment, the first thing you must do is to get through the regular defensive wall which surrounds these patients. They are like clams. They have been told they are no good so often, that they retire into a shell. You cannot do anything with them until you get them opened up. It will take a certain number of hours to get through the shell. But when you get through it, you can get them working with you. I do not like to emphasize the psychic element, but, of course, it is of value. There is, however, nothing mystical about the treatment. You can show the patients that they are getting better. It is not right to say that they do not want to get well. If you give them anything to hang on, they will stick. You will have to re-educate them mentally and physically, raise their threshold of resistance, and increase their self-control. It may take a little while, but just pull a few of these people out of the hole so that they are able to go about in their daily life, and you will see that they are mighty grateful patients.

A NEW METHOD OF SHOWING SOFT PARTS IN THE ABDOMEN BY MEANS OF THE ROENTGEN RAYS.

BY DR. WILLIAM H. STEWART,

NEW YORK, N. Y.

We are still seeking refined methods of diagnosis by means of the X-rays. Up to the present time, in order to visualize the organs below the diaphragm, we have been placing substances opaque to the X-ray within the gastro-intestinal tract. Now we have a new method of outlining the abdominal contents, especially the solid organs.

We have known for many years that the presence of air or gas increases the Roentgen detail of soft parts, but, so far as we know, in this country this fact has never been taken advantage of in bringing out details of the abdominal organs.

Our attention was called to this work by a case of gunshot wound of the chest referred by Dr. Willy Meyer. In attempting to localize the bullet great difficulty was encountered in ascertaining whether the projectile was *in* or *beneath* the diaphragm, or embedded in the lower lobe of the lung. In the discussion of this case before the New York Thoracic Society, Dr. Howard Lilienthal of New York suggested that we could rule out the question of the bullet being in the lower lobe of the lung by producing an artificial pneumothorax; if it was in the lung, it would recede from the diaphragm with the pulmonary collapse. Acting upon the suggestion, we saw no reason why the question of whether or not the bullet was below the diaphragm, could not be determined by producing a pneumoperitoneum, in which case we felt confident the liver would be clearly separated from the diaphragm. Dr. Arthur Stein called our attention to an article published in the November, 1918, number of the *Münch. Med. Wochenschrift*, by Dr. C. Goetze, in which he stated that the roentgenographic examination of the abdominal contents after inflation of the peritoneum with oxygen gave great increase of detail of the soft parts.

With these facts in mind and with the support of Dr. Stein, who inflated all of the cases in our series, we determined to learn the practical value of the method. The results, as will be shown

by the lantern slides, were somewhat startling. It was found that we were able to obtain clear detail of the liver and spleen, and that these organs were distinctly separated from the diaphragm, thus proving the value of the method in localizing projectiles or inflammatory conditions beneath the diaphragm. In addition, we learned that clear shadows of the other organs, such as the kidneys and pelvic organs, could be obtained. Intraperitoneal adhesions were easily shown and, in one case, a chain of enlarged mesenteric glands was demonstrated.

It is evident that a new field for investigation has been opened up, especially in obscure abdominal cases where the pathology involves the parenchymatous organs, and in subdiaphragmatic conditions which are so difficult to diagnose with the ordinary methods.

The technique employed is most simple. After the patient has been prepared for Roentgen examination, by a cleansing of the bowels and emptying of the bladder, he is put upon his back and the peritoneal cavity inflated with three or four liters of oxygen, the quantity depending upon the relaxation of the abdominal walls. The outfit required for this procedure consists of an oxygen tank, to which is attached at its outlet a small rubber tube of sufficient length. After the skin has been sterilized with iodine and anesthetized with Ethyl-Chlorid spray, an ordinary lumbar puncture needle is introduced through the skin, down to the fascia; it is then gently and slowly pushed on until it enters the peritoneal cavity—anyone who has had surgical experience can readily determine when this point has been reached—the shaft of the needle is then removed and the free end of the rubber tube attached. Oxygen is allowed to flow slowly into the peritoneal cavity until the abdomen has become more or less dome-shaped, when the tube is disconnected and the needle removed, the puncture being covered with an adhesive strip. The most desirable point of entrance with the needle has been found to be two or three fingers' breadth to the left of the umbilicus and about one and one-half inches below it. If abdominal scars are present it is best not to enter near same on account of the possibility of adhesions. The oxygen, as a rule, is absorbed in twenty-four to forty-eight hours.

Some patients complain of pain from the distension; this can be readily controlled by opiates. Most of them, however, suffer

no unpleasant symptoms. In all of our series we have experienced no difficulties. The danger of infection can be controlled by proper aseptic precautions.

Properly performed, we do not feel that there is any liability of puncturing the intestines. It has been demonstrated in living rabbits that the intestines will recede before a sharp body while, as soon as death occurs, the gut will be perforated.

222 West 79th Street.

DISCUSSIONS.

QUESTION: I should like to ask Doctor Stewart whether the danger of puncture would not be increased in cases of marked tympanites where the bowels are distended and in close contact with the abdominal walls.

DR. STEWART: I should hesitate to use the method under such conditions. In post-operative cases it would be well to keep away from the operative field.

QUESTION: What is the advantage of oxygen? Would not ordinary air do as well?

DR. STEWART: Oxygen is more readily absorbed and is always at hand compressed in tanks. We do not know of any objection to the use of air.

QUESTION: Is the oxygen injected under great pressure?

DR. STEWART: Oxygen in tanks such as we have on hand for anesthesia is used. It is under pressure, but can be controlled. In one of our earlier cases we produced too much distension and there was a leakage of oxygen through the puncture out into the extra-peritoneal structures; this interfered with X-ray details. It was absorbed in twenty-four hours, however, with no bad effects to the patient.

DR. STEWART: I neglected to say that it is not necessary to make the X-ray examination immediately after inflation. It is sometimes best to wait an hour or so, until the patient is quiet and accustomed to the distension.

INTESTINAL PROTOZOAN INFECTIONS IN UNITED STATES ARMY TROOPS.

BY MAJOR CHARLES A. KOFOID,
Sanitary Corps, U. S. A.,

BERKELEY, CALIFORNIA.

Mr. President and Members of the American Gastro-Enterological Association: It is a great privilege to speak to you today, not, however, from the standpoint of a medical man, but from that of a biologist who has been a student of parasitic forms of life for many years, and has been impressed by the frequency and importance of parasitic infections in animals generally. To correlate these observations and form conclusions applicable to man, we must look at the human parasitic infections, not as of recent origin, but as a contribution from the past, and as coming into existence with the evolving human race itself. This conclusion rests upon the fact that there is scarcely a single genus of protozoan parasites in man that has not been found in some or in nearly all the representative classes of the vertebrate series of animals. Thus the close relatives of the amoebae and flagellates that occur in man are to be found in other mammals. These amoebae and flagellates have come down to us from the past, and in the other vertebrates, some of these parasites are plainly pathogenic to their hosts, as, for example, *Giardia microti* to its host, the meadow mouse. To an inexperienced person, even, the lesions with which this flagellate is associated are perfectly evident upon careful examination. This parasite of the meadow mouse is closely related to a common flagellate parasite of man known as *Giardia intestinalis* (or *Lambia intestinalis* of older text-books). Fantham and Porter, at the Liverpool School of Tropical Medicine, found it in cases of trench diarrhea returning from Gallipoli and Flanders. Another flagellate in birds, related to the *Trichomonas intestinalis* of man, is abundant in the diseased tissues in the fatal "blackhead" of turkeys.

The work that I here present has been done in the Army Laboratory at the Port of Embarkation, New York, in part under a grant from the National Research Council for an investigation of the intestinal protozoan infections in man. It is based upon

stool examinations of two thousand three hundred men from overseas, and five hundred seventy-six home service men. The results show what might be expected. These protozoan infections of man, which are racial and are found to some extent in all countries, are increased in frequency and intensity wherever they do occur, by lack of sanitation, by crowding and by resulting contacts. War interferes with sanitation, increases crowding, and affords more abundant opportunities for contact. It is to be expected, therefore, that it would increase both the degree and number of these protozoan infections. To the Western Front, there came men from all parts of the world—from the tropics, as well as from the temperate zone. The men from tropical countries have brought their widely prevalent tropical infections with them. But we should not think that these infections are confined to the tropics. They are merely more prevalent, and perhaps more severe there, than in cooler climates. We are likely to get the impression, for instance, from the prevalence of amoebic dysentery in the tropics, that it is primarily a tropical disease, and dysenteric in its pathological aspects. This disease should really be called amebiasis, for it may be neither tropical in occurrence nor dysenteric in symptomology. We have found a large number of men who were not dysenterics when examined and who, in a large percentage of the cases, had had no record of dysentery, but they were carriers of the amoeba which may produce dysentery in some stage of the infection. The detection of these infections has not been made on the active stage of the amoeba but on the cyst or protected stage in most cases. These men are carriers of the infection and show none of the pronounced clinical symptoms of amebiasis, though mainly convalescents (in the overseas group) from various causes and below par in general health.

That brings me to another thing, and that is that the carrier problem in these infections is a very large one. Many of these men have no history of dysentery beyond a brief period of intestinal disturbance of unknown nature, generally not severe enough to result in treatment.

There are grounds in our findings and those of other workers, for the view that the proportion of carriers among cases of infection by intestinal protozoans is high. Whether or not it is higher, as it seems to be, than in the bacterial infections, such as typhoid

and diphtheria, must await fuller knowledge resulting from surveys of the normal civil population.

There is also a possibility that we deal in amoebic infections with races or types of different degrees of pathogenicity within the species as in the case of infections by pneumococcus. There is some evidence that there are distinct size races among the cysts of *Endamoeba dysenteriae*. We have no critical evidence as yet, however, as to any differences in symptoms accompanying infections by these several size races of this pathogenic amoeba.

Our statistical findings at the Army Laboratory at Debarkation Hospital No. 3, New York City, N. Y., are based generally on a single stool examination on men in transit through the hospital. In both the worm and protozoan examinations the percentages of infections detected would have been raised by several successive examinations above those reported in the table. Six successive protozoan examinations might be expected, according to our own experience, and the findings of Dobell in England, to at least double, and perhaps treble the percentage of infections detected. This rests upon the fact that the cysts in the stools come in successive but irregular waves of frequency. In our experience there may be six billion cysts of *Giardia* per stool on Wednesday and none found on an equivalent examination on Saturday. Cysts of the amoebas also vary greatly in times and frequency of appearance.

The home service men reported in the table were mainly men of the Medical Corps and food handlers. The proportion of men of foreign birth and of Florida negroes was relatively high among them. This group is, therefore, hardly representative of our troops before going overseas. From their sources above indicated it might be expected that the degree of infection would be higher than in the citizen soldiery or in the civil population as a whole.

For this reason the differences in the percentages of infections detected in the two groups of men examined are less striking than they would have been had the home service group been drawn from the same personnel as the overseas group.

An examination of the table shows the following significant results:

(1) The percentage of men with parasitic intestinal infections of one or more kinds in the overseas group is 66.9 per cent. as compared with 57.8 per cent. in the home service group, an increase of 16 per cent.

(2) The higher percentage of hookworm infections in the overseas group (6.9 per cent.) as compared with the home service group (3.8 per cent.) is due to the greater proportion of southern men in the former.

(3) The infection by whipworm, *Trichuris trichiura*, is extraordinarily high, 5.9 per cent., in the overseas men. The home service group, including many men from the south of Europe and from Florida, in both of which regions this parasite is rather common, had only 2.4 per cent. infection or less than half that of the overseas men. The contrast is even better shown in the data from 501,472 examinations made in army camps and hospitals prior to overseas service. In these there were only 1,975 cases of *Trichuris* infection or 0.39 per cent. as compared with 5.9 per cent. in the 2,300 overseas men. This is an increase of fifteen-fold as the result of overseas service.

The method of infection in whipworm is direct. The ova are highly resistant and remain viable for a long time in the feces or in soil or water contaminated by feces. The degree of infection in rural France is reported by Brumpt to be relatively high. The purification of water by chlorination does not destroy the ova of most parasitic worms. The ova are small enough to be carried by flies from feces to food. This great increase of infection by whipworm in the overseas troops means fecal contamination of food or water or both. Conditions on the Western Front during the rapid advances were such as to conduce to such contamination of food and water. It is quite possible that the 5.5 fold increase in infection by *Ascaris* in the overseas troops as compared with home service men is due to the same conditions. Infections by tapeworms which are not conveyed by such contamination show no such increase in overseas men.

The protozoan* infections of man detected by our examinations fall into three classes, the rhizopods including the amoebas, the flagellates including *Trichomonas*, *Tricercomonas*, *Embado-monas*, *Chilomastix* and *Giardia*, and the Sporozoa, including *Isospora* and other forms related to *Coccidium*.

The protozoan₁ infections of man detected by our examinations—Four of these are relatively rare, including *Endamoeba gingivalis* from the gums and tonsils, associated with pyorrhea; *Dientamoeba fragilis*, a non-pathogenic species reported recently in England and found only in the unencysted phase; a water amoeba

generally called *Amoeba limax* whose cysts appear to pass through the intestine unchanged; and *Chlamydomphrys stercorea* (not included in the table), a rhizopod with a bowl-shaped house or shell.

The common amoebae are three in number, *Endamoeba coli*, *Endamoeba nana*, and *Endamoeba dysenteriae*. The first two are reputed to be non-pathogenic, the last is the etiological agent of amoebiasis. All three are more abundant in overseas than in home service troops and unequally so. *Endamoeba coli* has 15.9 per cent. in home service men to 20.5 per cent. in those from overseas, an increase of 29 per cent. *Endamoeba nana*, a species long overlooked in man and described in 1917 by Wenyon and O'Connor from Egypt, proves to be the most frequent species in troops, both overseas and home service, examined by us, 29.3 per cent. and 27.8 per cent., respectively.

The greatest contrast appears in the case of *Endamoeba dysenteriae* where overseas men showed 12.8 per cent. to 4.3 per cent. in the home service group, a three-fold increase. Had it been possible to make six consecutive examinations of these men the percentage might have been doubled or trebled. This marked increase in this infection is comparable with the increase detected by British investigators in soldiers returning from the front and with the general increase of amoebiasis in the French troops and in the civil population of France as reported by Ravaut and others.

Another phase of this increase of parasitism in troops resulting from the war appears in the fact that the average number of infections in 2,144 overseas men was 1.6 per man to 1.4 in 559 home service men, an increase of 14.3 per cent. It was also apparent in a general way that the infections were more intense in the overseas men. It thus appears that the war tended to increase the amoebic infections noticeably.

The intestinal flagellate infections detected by us include five species. Of these three are relatively rare or easily escape detection, namely, *Trichomonas intestinalis*, which occurs in small numbers in fluid stools and whose spherical cysts might readily be confused with those of the smaller amoebae; secondly, *Tricercomonas intestinalis*, described by Wenyon and O'Connor in 1917, from Egypt, with elongated ellipsoidal four-nucleated cysts, and, thirdly, *Embadomonas intestinalis*, also described by the authors cited, as *Waskia intestinalis*, with slender pear-shaped cysts.

These three species were all found in free form in fluid stools though cysts of the last two were also found. Evidences of pathogenicity were lacking in all three species, though the first named is rather widely reputed to be a cause of flagellate diarrhea. It has undoubtedly been confused frequently with *Chilomastix mesnili*, which in our experience is twenty-fold more frequently met with in stool examinations than is *Trichomonas*.

The two most abundant human intestinal flagellates are *Chilomastix mesnili* and *Giardia intestinalis*. The former was found in 4.2 per cent. of the overseas men and in 3.5 per cent. of the home service group, an increase of 20 per cent. in the overseas men. This is represented in the stools by free flagellates and by cysts, the latter often in great numbers. The cysts are subspheroidal or pear-shaped with a stout, blunt, contracted end. The free flagellates have a short undulating membrane within the laterally located cytostome which simulates that of *Trichomonas* but is much shorter and has no free end. This organ has given rise to the misinterpretation of this flagellate as *Trichomonas* by clinical microscopists unfamiliar with *Chilomastix*. Its cysts also have been described as those of *Trichomonas* by Lynch. It is quite probable that clinical records of *Cercomonas* in medical literature refer in most cases to *Chilomastix*, as *Cercomonas* is not an intestinal but a coprophilic flagellate, and hence not a parasite.

Chilomastix has been described by Chalmers and Pekkola as the etiological factor in chronic diarrhea in the Sudan at Khartoum. The stools in which it occurs in the free state are often fluid or semifluid and of a diarrhea aspect and odor.

Giardia intestinalis, known in the older text-books as *Lambli*a, is the most abundant human intestinal flagellate. It occurred in 5.7 per cent. of the overseas men and 6.4 per cent. of the home service group. There was no increase in this infection detected in the overseas men. The spores are ellipsoidal, very transparent, usually binucleate, and are present in great numbers at times. The free forms are binucleate, are pear-shaped and have eight flagella.

This flagellate is often present in children, and in adults in cases of gastric or duodenal ulcer. It is the etiological factor in trench diarrhea from Gallipoli and Flanders, according to Fantam and Porter, and appears frequently in the clinical literature of flagellate diarrhea. It is quite intermittent in its appearances in the stool. In one case under observation for forty-two con-

secutive days, it was detected on only twenty-six days, appearing in recurrent waves that died out to reappear again and again in varying amplitudes.

It seems probable that the species of *Giardia* normal to rodents differ from those in man, but rats and mice may become facultative hosts to the *Giardia* of man and might thus spread the infection.

The Sporozoa of human intestine as we have found them, are light infections without marked evidence of pathogenicity. The infections detected by us were nearly twice as numerous in the home service as they were in the overseas men, but the numbers are too small to be of significance.

The most abundant human intestinal parasite is undoubtedly *Blastocystis hominis*, a yeast-like organism of uncertain relationships. It was first described by Prowazek as the spore of *Trichomonas*, but in reality has no proven relation to flagellates. Its size, shape, and appearance are superficially like those of amoeba in some of its phases, but it can be distinguished by its central, hyaline, usually non-nucleated, spheroidal, homogeneous mass, surrounded by a more or less granular zone, staining pink in iodine-eosin. This in turn may be enveloped in a perfectly transparent mucus sheath of variable thickness detected only by its exclusion of bacteria.

Blastocystis is usually present in cases of amoebic and flagellate infections and its presence and relative abundance appear to be a fair index of the state of the bowel. It is almost always present with infections of *Endamoeba dysenteriae*.

Another organism of undetermined nature is a spheroidal non-nucleated, homogeneous, often brownish spore of the same general size and appearance as the cysts of *Endamoeba coli*. This spore has often a slight protuberance, suggesting budding on one side and a central vacuole which often attends the origin of a hypha from a chlamydospore in the phycomycetes. We have interpreted this infection accordingly as that of an as yet undescribed phycomycete of the human digestive tract. It is included with the so-called "iodine-cyst" of Wenyon and O'Connor, the remaining content of this term being glycogen-bearing amoeba cysts. This phycomycete appears to have the distribution of a normal but rare parasite and not that of an introduced contaminative spore. It occurred in 8.4 per cent. of the overseas and 9.8 per cent. of the

TABULAR SUMMARY OF INFECTIONS BY INTESTINAL PARASITES IN 2,300 OVERSEAS AND 576 HOME SERVICE TROOPS OF THE U. S. ARMY AT DEBARKATION HOSPITAL No. 3, NEW YORK CITY, N. Y.

CASES OF INFECTION.

	Cestoda.			Nematoda.				Rhizopoda.						Flagellata.						Miscella- neous.				
	Total.	Negative.	Positive.	Dibothrioceph- alus Latus.	Hymenolepis Nana.	Taenia Sag- inata.	Hookworm.	Trichuris Trichinura.	Ascaris Lum- bricoides.	Endamoeba Coll.	Endamoeba Nana.	Endamoeba Dysenteriae.	Endamoeba Gingivalis.	Dientamoeba Fragilis.	Amoeba Limax.	Trichomonas Intestinalis.	Tricercomonas Intestinalis.	Embadomonas Intestinalis.	Chilomastix Mesnili.	Giardia Intestinalis.	Sporozoa.	Blastocystis Hominis.	Phycomycete Spore.	
Overseas.....	2300	763	1537	0	10	0	160	136	26	473	675	297	1	1	1	3	3	3	4	97	131	7	784	194
Home Service	576	243	333	1	3	2	22	14	1	92	161	25	1	1	1	1	3	1	4	20	37	4	181	57

PERCENTAGES OF INFECTION.

Overseas.....	2300	33.1	66.9	.0	0.4	.0	6.9	5.9	1.1	20.5	29.3	12.8	0.1	0.1	0.1	0.1	0.1	0.2	0.2	4.2	5.7	0.3	34.1	8.4
Home Service	576	42.2	57.8	0.2	0.5	0.3	3.8	2.4	0.2	15.9	27.8	4.3	0.2	0.2	0.2	0.2	0.5	0.2	0.7	3.5	6.4	0.7	31.4	9.8

home service men, but never abundantly in any case. Usually only several spores would be found on a slide.

The statistics of infections here detailed suggest that returning troops are bringing back from Europe increased infections with intestinal parasites, especially whipworm and *Endamoeba dysenteriae*. They also indicate the wide prevalence of carriers of these infections in the normal population. They should be looked for as complicating and accessory, if not primary factors, in obscure cases of intestinal and nervous nature.

DISCUSSIONS.

DR. B. B. VINCENT LYON, Philadelphia: I desire to apologize immediately for being on the program to open the discussion on this very interesting subject, first because of the fact that I have had no preliminary information, even in abstract form, of the substance of Major Kopoid's communication, and secondly because I believe that the opportunity to open the discussion was given to me merely as a courtesy on the part of Dr. Frank Smithies in order that I might call the attention of this section to a new method of investigation and treatment of diseases of the biliary tract. Therefore, with the knowledge of Dr. Smithies, and with the permission of President Bastedo, I believe it will be more profitable to make some remarks on a somewhat allied subject—catarrhal jaundice.

I had the opportunity of studying and treating by a new method sixteen cases of catarrhal jaundice, occurring in the medical service at Naval Base Hospital No. 5, Brest, France.

Most cases of catarrhal jaundice are due to ascending infection or extension of catarrhal swelling from a duodenitis. In addition, I have felt for some time, that the primary source of infection for both of these conditions is, in most cases, derived from infections of the gums, teeth, tonsils, nasal sinuses or the bronchial tree. This seems to be borne out in a study of these sixteen cases. All occurred within a relatively short period during the fall and early winter of 1918, when the epidemic of influenza was raging. Seven of these cases were serving in the fire or engine room on patrolling destroyers; all had recent bronchitis or rhinitis. Naval discipline prevented these men from expectorating below decks, the result being swallowing of infected saliva. Five cases followed attacks of influenza, with broncho-pneumonia; two cases followed operation for removal of nasal spur, and the last case was one of chlorine gas poisoning with its attendant coryza and bronchitis.

The whole series of sixteen cases, therefore, had infections of the mouth or air passages and were swallowing infected material resulting in gastric and duodenal infection or catarrhal states, which eventually spread to and occluded the common bile duct. In the last seven cases seen and studied the material aspirated from the duodenal zone

showed cultural identity with the bacteria isolated from the mouth or air passages.

The first nine cases were treated by the usual symptomatic, or "expectant" method of bed rest, modified diet, calomel, sodium phosphate and hexamethylenamine, etc. The average duration of jaundice in this first group of nine cases was thirty-five days.

The second group of seven cases were treated, in addition to the symptomatic plan, by a method of direct disinfection of the stomach, duodenum and biliary tract. Throats were gargled with strong potassium permanganate solutions, a duodenal tube was then passed and the twelve-hour fasting stomach rinsed and disinfected. The tube was then allowed to pass into the duodenum, which was also rinsed and disinfected, and then douched with a 25 per cent. solution of magnesium sulphate.

The use of the latter was the result of my previous experiences in the examination of the biliary tract, following some experimental work of Dr. Meltzer, of the Rockefeller Institute, the results of which he published in the spring of 1917. He found that by rinsing the duodenum with such a solution of magnesium sulphate that the sphincter of the common bile duct would relax and that shortly afterwards the gall-bladder would contract and expell its contents into the duodenum. This I had previously verified clinically and found that it worked well in the treatment of catarrhal jaundice by serving to uncork the plugged-up common bile duct. The direct proof of this lay in the fact of promptly restoring biliary drainage and shortening the duration of jaundice in this second group of seven cases to an average of seventeen days, a gain of a little over 50 per cent.

I feel convinced that this method is a valuable one and expect shortly to publish in the *Journal of the American Medical Association* the details of my investigations during the past two years. I hope that it may prove to advance our ability to scientifically and clinically diagnose cholelithiasis, cholecystitis, choledochitis and chronic pancreatitis and that it may open an avenue of efficient and direct medical treatment of several of these conditions.

DR. ARTHUR F. CHACE, New York City: We are greatly indebted to Major Kopoid for his comprehensive presentation of the subject of protozoal infection. Four years ago, routine examinations of feces were made from the medical service of one of the large New York hospitals. These examinations showed the frequency of protozoal infection. The results of this investigation were reported in the Section of Pharmacology and Therapeutics of the American Medical Association three years ago. Cases of cercomonas and of lamblia responded promptly to the methylene blue treatment. The examination of feces in obscure cases of diarrhea should be made for protozoal infection.

DR. SEALE HARRIS, Birmingham, Ala.: I think that it is very timely, when large numbers of army troops are returning to consider this

subject; for I have no doubt that these men will be found to be carriers of many infections that have not been previously discovered in this country. As to their not having had any histories of trouble, I should like to say that most of the men that have been about in France have had some diarrheal trouble. It was particularly bad after the drive against the Germans. There were epidemics of dysentery in the American, as well as the French and British armies. It was usually very mild, but extended as far as Paris. It was found after the cases were studied bacteriologically and microscopically, that a large portion of them were of the bacillary type; but the *entamoeba dysenteriae* was found in a number of cases by Dr. Siler, of the American Expeditionary Force. The Germans in their retreat seemed to have made an effort to ruin everything when they left. They defecated on chairs and tables, and around wells; and from these visible evidences, I judge that they must have had a great deal of dysentery among them. The African troops that were there also suffered in this way. After the Chateau Thierry drive, the Germans left dead horses on the field for days. Flies were everywhere, and they could not be kept off the food.

It is a routine with me to make an examination of the feces. The gastro-enterologist who does not make a routine of examining the feces is not examining his patient thoroughly. I, too, have found the *Cercomonas* frequent; and in many cases, these patients had had no diarrhea at any time. I found, in a number of cases, that there was achylia present; and I gave the hydrochloric acid. It did improve the diarrhea.

MAJOR CHARLES A. KOFOID, New York (closing): With regard to the cure of these flagellate infections, I can speak only from a limited observation and a review of the recent literature on the subject abroad. I think that it is an entire mistake to regard a case as cured that has not been under observation for many months with repeated stool examinations. These organisms habitually, even in many treated cases, run in cycles of a certain number of days or weeks. They will come and go. You may have six billion spores of *Giardia* on one day, for instance; and three days later none. Two or three weeks later, they may be back again, however. They come and go. It is unsafe to feel that you have cured the case by washing the intestine out. You have relieved the condition, but it may come back.

A few negative examinations do not establish a cure. The British War Office, in a recent bulletin, quoted the statement that cases of amoebiasis complicated with flagellate infections are incurable. The original French observer had, however, used only five days' combined treatment with bismuth emetin iodide and subcutaneous emetin, instead of twelve, as the British recommended. I think, therefore, that their conclusions are not to be accepted until based on cases which have been treated for twelve days.

The cases of flagellate infection do not yield to any known specific.

You may have palliative treatment with saline, iodine, or turpentine and rid the bowel of them temporarily, but they may come back. I have attempted to treat giardiasis in rats, which is the equivalent of this flagellate infection in man, with arseno-benzol intravenously. Rats thus treated were at once rid of the spores of the flagellate in the stools, and they stayed rid of them for five weeks. Then, owing to military exigencies, I could not examine them again for six months. The treated rats were without spores then; and at autopsy, none that had had the arseno-benzol treatment showed the infection.

Griffon and Roux have also used arseno-benzol for *Giardia* with promising results; so I hope that those who have these cases to deal with will try the arsenic preparations. The French are using the substitutes for arseno-benzol, as well as the drug itself.

There is no question but that the *Chilomastix* is widely distributed and common in the United States, and that it very often occurs with amoebic infections. That it is a very widespread human parasite at times, abundant when present, and exceedingly persistent in its hold upon the host, as well as very erratic in its occurrence in the stools, is certain.

The conditions in France and England regarding these infections that the troops are bringing home are much the same as here. We may expect that an invasion of these parasitic infections will follow the war in this country, as they followed the Philippine War here. You may have cases of liver abscess, lung abscess, brain abscess, etc., in which these amoebae will be found to be the cause of the lesion; and you will find enlargement of the spleen, symptoms referable to the appendix, obscure skin infections and other conditions that may be attributed to this organism of amoebiasis.

Ravaut, a recent writer in France, states that amoebic infections have increased rapidly in that country during the war, and are traceable in some instances to Colonial troops. He notes two things about the patients as striking; that they are usually not dysenterics, and that the diagnosis of amoebiasis is too often made on the autopsy table. He suggests to physicians that they acquaint themselves with the varying conditions of this infection and be not surprised when they meet it.

¹Figures and descriptions of the cysts of protozoans occurring in human feces will be found in *Criteria "for Distinguishing the Endamoeba of Amebiasis from other Organisms,"* by Kofoid, Kornhauser and Swezy, *Arch. Internal Med.*, Vol. 24, pp. 35-50, July, 1919.

A BRIEF EXPERIENCE WITH APPENDICOSTOMY AND
CECOSTOMY FOR INTESTINAL STASIS IN
EPILEPSY AND NEURASTHENIA.

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I have had an opportunity in the last year or two to observe a small group of patients, two with neurasthenic symptoms, two with epilepsy, who had a considerable degree of intestinal stasis and in whom appendicostomy or cecostomy was done with subsequent washing out of the large intestine for a period of six to twenty-six months. The operation was done on the theory that delay in the passage of the contents of the colon and possible absorption of toxic material was responsible for some or all of these symptoms and that the patient would be relieved by keeping the bowel well cleaned out.

The patients had all had a long thorough course of medical treatment under the best conditions, some of them had stayed for a year or more at one of our best sanitariums with little or no improvement of symptoms and the operation was only done as a last resort. They were all patients seen in consultation with other physicians and had been considerably encouraged by other physicians, who had seen them, to have the operation performed. Personally I did not have much enthusiasm for the method, but I was placed in a fortunate position to be able to follow the cases and to observe the results of the operation and subsequent treatment.

The literature of the subject is rather scanty and confusing. We are very likely to find immediate good results reported and the late results not given. Naturally the late results are chiefly interesting.

These cases, though few in number, are reported largely to stimulate discussion and bring out the experience of other men at this meeting. It seems worth while to have the facts in these cases even if the results are not brilliant.

SYMPTOMS AND SIGNS.

The two epileptic cases gave a typical history of epileptic attacks of long duration, namely 14 years. The other two cases had pronounced chronic neurasthenic symptoms, headaches, insomnia, attacks of indigestion, anorexia, loss of weight, poor circulation, fatigue, mental apathy, bad taste in the mouth, abdominal gas and occasional tenderness, some staining of the skin, etc. They were all habitually constipated and had a palpable "juicy" cecum, and X-ray examination showed considerable delay in the passage of barium and food material through the large intestine, so that at the end of two days there was still a good-sized residue throughout the colon. All were slow in emptying the right side of the colon. We realize that intestinal stasis is not a perfectly definite diagnosis; that the normal bowel schedule varies a good deal; that individuals may be in apparent perfect health and have very different rates of emptying the colon. We also know that there is a good deal of normal variation in the same patient at different times. These patients, however, emptied the large intestine habitually at a much slower rate than the average normal person.

SELECTION OF CASES.

It is a difficult matter to select proper cases of this type for operation when we are dealing with neurasthenic or neuro-mental or epileptic patients who are also constipated. We have a good deal of difficulty in deciding which condition is primary—in short, whether they are constipated, because they are weak, neurotic and unstable and have low intestinal tone, due to fatigue, worry and distress; or whether the reverse is true, namely, that they have neuro-mental symptoms because they are constipated and are being more or less poisoned by intestinal absorption; or whether possibly we have a vicious circle which it is wise to break at whatever point we can.

We must emphasize once more that a long thorough medical treatment under the best conditions should precede any thought of surgical methods. This medical treatment will include rest cure, psychotherapy, exercises, diet, the use of oil, agar and suitable drugs.

Since it is difficult to pick out the neurasthenic or epileptic whom we feel sure that intestinal surgery will help, we must, of

course, condemn the indiscriminate use of surgery in such cases. The cause of epilepsy in particular is very complex. Many of the factors which are believed to cause epilepsy are not peculiar to it. We commonly find everyone of the lesions which are associated with epilepsy in other people without epileptic symptoms, and while abnormal conditions of the bowel are probably important in causing attacks in *some* epileptics we have no evidence that this is true of *all* epileptics. The X-ray often shows changes in the intestinal tract in epileptics like those found frequently in non-epileptic. We find intestinal stasis, ptosis, etc., but we must remember that while all epileptics are constipated, very few constipated people have epilepsy.

Many methods of relieving epilepsy have been used at various times in the world's history and later discarded as useless. Is this another?

Other things being equal, the greater the delay in emptying the colon, the more suitable these cases are for surgery, but colon stasis is far from being the only factor to consider. The operation of cecostomy or appendicostomy is probably justified in carefully selected cases but it is largely experimental at present and I believe this should be explained to the patient.

OPERATION.

We shall not discuss at length the kind of operation best suited to such patients. We know that only a fraction of one per cent. of cases of intestinal stasis are obstructive or organic, the rank and file are functional, atonic and ptotic, and, therefore, appendicostomy or cecostomy was chosen for these patients in preference to colectomy because they are much simpler operations and more suited to the simpler type of non-obstructive case. They are free from the objections of the short circuiting operations and are far less serious than a right colectomy.

There are some purely local after effects of this type of operation which may be important in neurotic and sensitive persons. There is often a disagreeable amount of fatigue and disability in a nervous woman from *any operation whatever*, which may last for weeks or months. In addition any colostomy may be disagreeable, there may be some local irritation, a little pus discharge about the wound, slight odors, some local pain at first so they cannot walk about, all of which are disagreeable to a

sensitive woman. The after effect must be good enough to overcome all these features.

RESULTS.

The actual results of the *operation* and subsequent lavage are difficult to judge in this class of patients; the firm belief of the patient that something definite is being done to help him has a marked effect, which is purely psychic, and we must also remember that a remission of epileptic attacks may follow *any* surgical operation of whatever sort.

The immediate effects of the operation were not important in most of the cases, the convalescence was reasonably prompt and easy. In one neurasthenic patient the immediate effects were trying. For several months after the operation she was very nervous and tired, and there was considerable local irritation from the wound which prevented walking about.

The late results have been as follows: Both neurasthenic patients considered the operation a real help; in one a definite improvement in symptoms, such as headache, dullness and fatigue, occurred; intestinal gas, formerly very troublesome, almost disappeared; sleep improved; attacks of depression disappeared. In the other neurasthenic case, while the operation was very simple and easy and convalescence rapid, the end result was not satisfactory. The patient had many nervous and mental symptoms and could not be said to be really improved at all in spite of the fact that the irrigation was very simple and easy and thorough, and in this patient, the mental condition was for a time precarious. After 10 months, the patient complained a good deal of discomfort in the neighborhood of the opening and also from the dressings, so the appendix was removed and the abdomen closed. Her mental condition has continued to improve slowly under careful sanitarium treatment.

The results in the epileptic cases were as follows. In the first patient (Case 3) who has also chronic nephritis and who had been having attacks three or four times a year, the attacks entirely stopped for a period of eight months after the operation. Since that time they have occurred again in a slightly milder form than before and rather more frequently than before, the two attacks in the last six months have been very mild. (See diagram.)

The other epileptic patient, a more serious case, in whom the

CASE 3—X REPRESENTS AN ATTACK.

Year	Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
1916	X	X	X
1917	X	XX	X	Operation
1918	X	X	X	X	X
1919	X	X X	X	X	X

attacks were frequent and severe before operation, averaging one to two a month, had no attacks for two months after operation, then the attacks continued just as often as before, though some were not as severe.

There have been no marked changes in physical signs in any of these cases, except that one patient gained 15 pounds. Two patients were recently examined to see if the action of the colon had become more prompt and satisfactory than before operation. We tried to judge of this by an X-ray examination and it was found that when examined at the end of 36 hours, the food and barium had made better progress through the intestine than before the operation and that the right side of the colon was much better emptied out. Both patients, however, were somewhat behind the average normal schedule for emptying the colon. Of course, the daily irrigation was omitted at the time these barium tests of motility were made. We have considered the *shape* of the bowel of little importance compared with its *function*. We agree with Cannon that "handsome is as handsome does."

I am left with somewhat mixed feelings at the end of these observations. All the patients feel they have been helped and are glad that the operation was performed. One neurasthenic case is definitely better now than before. This is not true of the other neurasthenic case.

The results in the epileptic cases were rather striking for a time, but we find the patients at the end of one to one and one-half years in a condition which is much like that before operation. In looking over the literature and talking with various men, it seems that we may expect an occasional brilliant result in such cases, perhaps one in ten, depending on the selection of patients; that toxemia will be removed; that changes in intestinal bacteriology will occur and the intestinal muscles and mucous membrane and secretion will be improved. On the other hand, in many or most such cases we shall get little or no result. In epilepsy we do not change the neurological make-up of the patient, we simply remove a contributing cause, not a primary one.

In Gant's¹ series of about two hundred cases of appendicostomy or cecostomy, chiefly for colon infections, only two cases of epilepsy were treated. They were relieved, but attacks recurred in two to six months. Brewster,² at the Massachusetts General Hospital, in a series of twelve similar cases of epilepsy and colon

stasis, using a bigger operation, right colectomy, reports that while the general condition of most of the cases improved and they were largely relieved of their constipation, there was slight, if any, change in the epileptic attacks. I have not found one considerable series of cases in the literature where uniform benefit has been reported by a reliable man. In talking with those in charge of the well-known colonies for epileptics in Massachusetts and New York, I do not find much enthusiasm for this kind of treatment. Their few epileptics with appendicostomy and daily lavage have not received any more benefit than might be expected from the use of ordinary irrigations at regular intervals.

It is difficult to decide *when to close the opening*; in two of our cases it was closed in six and ten months respectively, because of local discomfort and because nothing seemed likely to be gained by keeping it open and continuing the irrigations longer. In the two other cases it remains open (January, 1920), 19 and 27 months after operation. It is, of course, possible to test the probable effect of closing the opening at any time by simply stopping the irrigations for a period and watching the result.

Since following this small group of cases, I have seen several patients with definite epilepsy associated with definite atonic delay in the colon and have hesitated to advise any operation on the colon. I have also several neuropathic persons under observation at present with marked degrees of non-obstructive intestinal stasis and even after long and only partially successful medical treatment I have hesitated to advise even the simple operations which we have been considering.

CONCLUSIONS.

In summing up we may say that all our patients were glad that the operation was done, one neurasthenic patient was definitely and permanently improved and one was not. In both epileptic cases the immediate results were fair or good (two to eight months without attacks). The late results were unsatisfactory. I have hesitated to advise operation in later similar cases in both groups.

Constipation is so frequent in neurasthenics and epileptics that we are tempted by the theory that their symptoms are due to intestinal stasis and toxemia. On the other hand, it is too much to expect that we can change the character of these patients by opera-

tion and lavage, without help on their part. We may remove a contributing cause but not a primary one.

We shall always have difficulty in selecting patients for such operations because we shall often be in doubt which condition is primary, the stasis or the nervous condition, and we usually advise operation to break into a vicious circle and to remove a contributing cause.

The question of when to close the colotomy is also difficult to decide.

We feel quite doubtful about extending surgical treatment to any non-obstructive colon stasis case (unless we have stasis of one hundred hours or more). If we do, the operation must not be risky and must give relief without troublesome complications. Such operations are largely experimental at present.

Case 1.—Miss M. A. D., 31; diagnosis neurasthenia, abdominal ptosis, marked colonic stasis without obstruction.

Mother neuropathic, one brother chronic alcoholism.

Pulmonary tuberculosis at sixteen which was arrested, average health, active, athletic until seven years ago, since then anorexia, epigastric distress or pain, occasional vomiting, frequent regurgitation of food, chronic constipation, headache, fatigue, insomnia, irritability and depression.

Physical Examination.—Tall, thin, nothing abnormal in heart and lungs, gastric secretion variable, marked abdominal atony and ptosis. Stomach reached four inches below the navel. The colon entirely below the navel. Stomach and small intestine emptying normally. Seventy-six hour uniform colonic stasis without obstruction found on several examinations. Feces no important changes.

Much sanitarium treatment for years, diet, rest, exercise, abdominal massage, oil, agar, with little improvement.

Appendicostomy, February, 1918, Dr. D. F. Jones, of Boston; rapid recovery from operation; irrigation of colon twice a day at first, later once a day for ten months; no important changes in nervous or digestive symptoms; mental condition precarious; somewhat stronger physically; steady gain in weight of fifteen pounds. Patient is glad the operation was done. At the end of ten months considerable discomfort near the opening, so the appendix was removed and the abdomen closed. Her mental condition has continued to improve slightly under sanitarium treatment (one and one-half years after operation, Dr. F. X. Dercum, Philadelphia).

CASE 2.—Miss E. L., 30; diagnosis, neurasthenia, ptosis, colon stasis, atonic type. Father and mother, two brothers and two sisters well; one sister chronic headaches, one brother mental.

Athletic, very active, repeated tonsillitis, tonsillectomy age four-

teen. Eight years fatigue, depression, insomnia, intestinal indigestion, constipation, distress, gas. Chronic appendix and uterine fibroid removed five years ago. No improvement of the digestive symptoms. Best weight one hundred thirty, loss to one hundred ten, gained again to one hundred twenty before operation.

Physical Examination.—Small, muscular; sallow, pigmented skin, gastric secretion below normal. Feces nothing abnormal. Stomach, slight ptosis (two inches), marked ptosis right half of colon, redundant hepatic flexure, moderate adhesions tip of cecum, no ileal stasis, marked forty-eight hour stasis in colon, not obstructive; tip of cecum remains filled after emptying ascending and transverse colon.

Much sanitarium treatment, rest, diet, exercise, abdominal massage, oil agar, enemata. Recent gain of ten pounds in weight, little change in nervous or digestive symptoms.

June, 1918, cecostomy, Dr. G. W. W. Brewster, Boston. Lavage daily for five months, then every day or two, and one year later every second day; stool only with irrigation; was very tired and nervous for several months after operation; also had local irritation in wound so she could not walk about very much for four or five months. Patient thinks the operation was worth while and has been glad to suffer the discomfort of it for the sake of what improvement has followed. Sleep is good. There is less fatigue, no depression. The head is clearer, which the patient considers "a great improvement." The patient is not strong but enjoys life and works quite steadily part of each day. No further gain in weight. X-ray examination of the bowel a year after operation shows right half of colon well filled twenty-four hours after barium meal, this is quite well cleared out by irrigation. Position and mobility of right colon same.

(Cecostomy still open nineteen months after operation, January, 1920.)

CASE 3.—Mr. W. B. P., 52, draftsman; epilepsy, chronic nephritis with hypertension, colonic stasis, was referred to me by Dr. W. G. Morgan, of Washington, after operation one and a half years ago. I have followed him for sixteen months.

Family history good, two healthy children, athletic, always constipated, piles. Epileptic attacks began fourteen years ago, at the age of thirty-seven, usually nocturnal, had two or three a year; strong, good sleep, no headache, treated by low proteid diet and bromides for years.

Well developed and nourished, good color; blood pressure, systolic 175 to 210, diastolic 105 to 125, pulse 82, three or four premature beats per minute, moderate enlargement of heart, systolic murmur over whole precordia, sulphonephthalein test of the kidney 55 per cent. in two hours. Urine 1,600 c.c., gravity 1,009, albumin one-eighth to one-fourth per cent., no sugar; scanty sediment, few hyalin and granular casts and renal cells.

X-ray examination showed that stomach and small intestine emptied normally, marked ptosis and spasm of the colon resulting in forty-eight marked cecal stasis, no adhesions.

Appendicostomy, October, 1917, daily lavage of colon, stopped work for one year, attacks (see Chart), formerly three a year, stopped entirely for eight months. In the following year there were nine, all mild, and in last six months two attacks both very mild. Feels well except for attacks. Bowels move normally with irrigation. Several recent X-ray examinations of the bowel nineteen months after operation (omitting irrigation) showed that the right half of the colon empties in twenty-four to thirty-six hours and the barium is scattered through the left half of the colon or is all in the rectum. This residue is almost entirely removed by subsequent irrigation. Slight gain in weight, blood pressure remains the same, has worked steadily for fifteen months as draftsman.

(Appendicostomy still open twenty-seven months after operation, January, 1920.)

CASE 4.—Mrs. B. S., age 26, married one year; diagnosis, epilepsy, ptosis, colon stasis.

Father and mother well, three sisters nervous, two brothers and one sister well. For twelve years constipation, daily laxatives, epigastric distress and gas, occasional headache, nine years epilepsy, usually nocturnal, one or two attacks a month, slight mental depression, memory poor, bromides for year, weight six years ago 124, now 110.

Poorly developed and nourished, neurotic, slightly stupid, irregular teeth, tonsils slightly enlarged, heart and lungs normal, cecum palpable and slightly tender, gurgles on palpation, stomach contents after test breakfast nothing abnormal, urine normal. X-ray examination showed marked ptosis of the stomach (5 inches), colon entirely below the crest of ileum, stomach and small intestine emptied in approximately normal time, colon well filled throughout, up to fifty hours.

Appendicostomy September, 1918, daily lavage for six months, attacks stopped for two months, then were just as frequent as before, some were milder, general condition improved somewhat, less depression and nervous symptoms, gain of five pounds, appendicostomy was allowed to close after six months.

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DISCUSSIONS.

DR. ARTHUR F. CHACE, New York City: Many cases of petit and epilepsy are undoubtedly due to intestinal putrefaction. It is wiser to eradicate the intestinal putrefaction by medical means than by surgical. It is more a question of disordered physiology than of surgical pathology. Few, if any, of these cases are improved by cecostomy. Better results are obtained by duodenal irrigations. A vicious cycle can often be broken up by duodenal irrigations of 4 per cent.

sodium sulphate solution. Through and through irrigation may be established by inserting a rectal tube.

DR. WILLY MEYER, New York: Dr. Weir, of New York, proposed this interesting operation about fifteen years ago; and since then I have done it quite frequently. When the appendix is opened during the operation to test its permeability, we may find the conditions that make its use for the flushing of the intestine impossible. Then cecostomy is in order. I have done appendicostomy quite often; and am enchanted with its results. Surgeons usually get these patients referred, not on account of constipation, but for too frequent movement of the bowels, colitis, mostly "ulcerative" colitis. I will not go into this, except to say that there is no better treatment for obstinate chronic ulcerative colitis than appendicostomy and colonic flushing. Even in tuberculous colitis, in patients with definite tuberculous ulcerations, I have seen what one might call a cure for three or four years; the trouble recurred later, because the patient had stopped the mechanical treatment.

I personally believe in the teaching of Sir Arbuthnot Lane, who brought out colectomy and short-circuiting in these cases. I think that it contains a great deal of truth. No doubt, the large intestine is the breeding place for many intractable symptoms; if we can take hold of them, the patient will be much benefited. Two years ago, at the meeting of the American Surgical Association in Boston, Dr. Bevan spoke most emphatically on this subject, and stated in his conclusions that he did not believe in the efficacy of this operation. I ventured to rise, in the discussion, and say, "What are we going to do with these cases? They complain and complain, and the most intelligent internists have tried in vain to relieve them. Let us think of appendicostomy." To my regret, I personally never got a case of stasis for operation since then. I was glad to hear that Dr. White has followed up the result of appendicostomy in neurasthenics. They certainly represent the worst class to take hold of in order to reach a conclusion on the value of this operation. I think that six to twelve months is by far too short a time in which to tell of results. These patients, who have been sick fifteen or twenty years, cannot be cured in such a short time. They must be treated for several years. If appendicostomy is properly carried out, it is no annoyance whatever to the patient. The principal point is to leave the appendix, in its entire length, just let the tip peep out above the skin. I always do a McBurney operation. The patients have a very small opening which does not discharge. Through it they easily introduce the soft rubber catheter every day. It is their "safety-valve," in the real sense of the word.

DR. G. W. MCCASKEY, Fort Wayne: I have been very much interested in Dr. White's report, and I heartily concur in his very conservative and, I think, very judicious conclusion. I have had no per-

sonal experience with cecostomy or appendicostomy in the treatment of epilepsy, but I have been impressed for years with the fact that a certain number of these cases that have come to my consulting room for treatment along the line of internal medicine have given a history of a gastrointestinal upset at about the time of the attack of epilepsy. In a considerable proportion of these cases, this gastrointestinal upset regularly preceded the epileptic seizure. Even though this is true, however, I think that we should not be willing to go so far as to say that in finding these conditions, we are finding the real ultimate cause of the epilepsy. We are finding what Dr. White judiciously terms contributory causes. There is a pathology back of these, about which we know very little today. The bacillus epilepticus has come and gone, without our being any the wiser because of it. With Dr. Meyer, I would not absolutely condemn this operation; and I think that anything that holds out hope to these patients is worth trying, especially as we so commonly find gastrointestinal putrefactions and intoxications. The operative treatment may temporarily relieve the patient, but the rule is that the condition will come back. It is notorious that almost any operation may make the patient free from epileptic seizures for some time—months, perhaps; but almost invariably the seizures return almost as badly as in Dr. White's cases. So I have personally, no confidence in the permanence of the results; because the real pathology is untouched and the line of treatment that will put the patient's gastrointestinal canal, etc., in the best possible condition is the best thing that we can use in the case of the epileptic. Whether we do operate or not, and endeavor to flush out the colon, I am inclined not to take a very enthusiastic view of it. We have seen epileptic seizures go on for years with all sorts of treatment, and we have seen them stop entirely.

A patient recently came under treatment, and the history was that an aunt of the patient had had these attacks for twenty years, after which they had stopped, and for years she had not had a single attack. That is not the rule, but it occurs more frequently than is generally believed.

My conclusion would be, let the gastroenterologist treat these cases from the standpoint of the gastro-intestinal pathology, and not assume that he is treating the underlying pathology of epilepsy.

DR. ALEXIUS MCGLANNAN, Baltimore: I have had opportunity to do this operation twice in cases of epilepsy. One patient was an epileptic man; and the other, a young woman. The man was not improved, any more than these cases were. He improved for a few months, and then went back. He improved again, when I closed the fistula, and then he went back, just as he did after the original operation. The woman was neurotic and had stasis. We did an appendicostomy, and washed out the colon. It did not improve her mental condition; and the open wound bothered her, although it did not discharge. We closed the wound, and she stayed in the asylum where she died, a year and a

half after we had closed the appendicostomy. So my results show that the operation did not help the patient, although I am sure that it did not hurt either one of them.

DR. SEALE HARRIS, Birmingham: I have been very much interested in Dr. White's report of these cases, for the reason that the operation of appendicostomy has been quite a popular operation in the South. A number of years ago it was heralded as a cure for amebic dysentery, and a great many of these cases were operated on at that time. I had the misfortune to see a number of cases that had had the appendicostomy done and had not obtained any relief from it.

The question of the cure of amebic dysentery is very simple, and the operation fell into disrepute—and, I think, justly so, from what I saw. In the first place, the operation of appendicostomy does the very thing that surgeons talk so much of in relation to the question of chronic appendicitis: It produces adhesions around the appendix and produces the symptoms of chronic appendicitis. Among the cases that I happen to know of, I do not know of a single instance in which the operation cured amebic dysentery, but I did see a number of cases in which the operation was performed without results, and later the wounds were closed.

It seems to me that this operation is a piece of empiricism that is hardly justified. Of course, epilepsy is a thing that we cannot do much for, any way. At the same time, I think that it must be in very rare cases that this operation is indicated. To resort to it in these cases is treating symptoms, without any effort to get at the real cause. The lavage of the colon through the appendix, when you attempt to cleanse out the colon thoroughly in that way, is rather a difficult procedure; and it is a question how much good it does. I notice that the doctor speaks of it as "a brief experience" with appendicostomy and cecostomy in intestinal stasis. Do not misunderstand me. I do not wish to criticise Dr. White for his report, but I wish to emphasize the fact that, judging from my experience and that of other men with amebic dysentery in the South, I believe that the briefer the experience with this operation, the better it will be for the patient and the physician.

DR. SEYMOUR BASCH, New York City: Even theoretically, I do not see how one can expect results from this operation, and certainly not in neurasthenics, as was said by previous speakers.

As regards epileptics, if epilepsy is an intoxication (and that is only one of the theories advanced to explain it), why make the intestinal opening so low down, after all the intoxication has been absorbed through the portal circulation?

I want to back up what Dr. Chace has said regarding lavage. I do not believe that we can prove that intestinal putrefaction is the cause of epilepsy, any more than you can prove that anything else is. If, however, you assume that intestinal intoxication is the cause, why not use simple duodenal lavage, instead of a major operation? It does

more than wash out the intestinal contents. It opens up the biliary passages and causes absorption through the duodenum and by the portal circulation. The lavage can be carried out in your office, and it causes the patient very little inconvenience or trouble. We have had enough, I think, of major surgery for indeterminate conditions. We see patients with all kinds of indefinite disabilities, and it is time that we stopped looking upon human life so lightly.

DR. FRANKLIN W. WHITE, Boston: This paper has produced the effect that I hoped it would, namely, to stimulate discussion and bring out the facts from various sources and make us more clear with regard to what we should do in such cases. I did not bring up the subject with the idea of advocating such operations, but merely to record results. It is certainly a wise suggestion to first make a trial of duodenal irrigation in these cases. Some people do not take kindly to a tube; but a little experience and kindness usually make it an easy affair. It is much milder than the operation, and can be stopped at any time.

With regard to the use of these operations in colitis, I wish to say that I avoided that subject because it was outside of the scope of the paper.

Regarding the treatment, I agree with Dr. Willy Meyer that it will have to be a long one. Two of these four patients still have the opening at the end of $1\frac{1}{2}$ to 2 years, and are still carrying out their lavage. When to stop is a difficult question to decide. The patients are beginning already to ask about this, and I should be glad of any suggestions from the members of this Association.

As to fixing the cecum, I feel as Dr. Stewart does, that the condition is not comparable to chronic appendicitis where adhesions may be disagreeable. Some of those loose sliding cecums are better for being fixed.

As for treating the symptoms without an effort at removal of the cause, you will remember that these two epileptics did not show any one of the many definite cerebral lesions or conditions which occur with epilepsy. They were of the group that the neurologist calls idiopathic. In this group, we cannot remove the cause, because we do not know what it is, and we are doing the best we can.

ADDRESS OF SIR WILLIAM ARBUTHNOT LANE.
(CHRONIC INTESTINAL STASIS.)

LONDON, ENGLAND.

Mr. President, Ladies and Gentlemen: I saw a note on this sheet of paper that I was to speak on some subject, but it did not give me an idea as to what subject I should speak on. I do not think that I can interest you more in any other than I can in the subject that your President has just mentioned, Chronic Intestinal Stasis.

Talking about intestinal stasis is like talking about many things. It is interesting to observe the difference in the attitude between British and American medical men towards it. When you start anything new in England, everyone thinks you are a boaster, and are unfit to be a member of the medical profession. In America, it is different. The American is glad to hear what you say. He is interested, goes back on his previous work, and does what he assumes to be a modification and improvement on your operation. After a little time, then, the American medical man drops it, and recently it pleased me very much to find that after the Americans had dropped my operation, the British had taken it up. They have grasped the situation, and in a way that, I think, is far better than you have. While for twenty years I had no one over there on my side, now everybody is doing the operation, not only for reasonable conditions, but also for unreasonable ones. They go to extremes.

I think that chronic intestinal stasis, as the cause of disease, acts by converting a food supply that in the normal subject is sterile, into one that is contaminated. This alters the relation of every organ in the body to the food supply, and produces as much of a change as is produced when you try to make a coal heaver out of an inexperienced person. The skeleton of the coal heaver is as different from ours as ours is from that of the apes. The kidney, liver and spleen are as much altered in chronic intestinal stasis; because they have to perform a new function. Originally, they worked with a sterile material; while now they have to make use of one that is foul and filthy. They, consequently, undergo changes, which you call disease, but which

are the result of the different behavior of these organs in different surroundings—just as the coal heaver or coal trimmer finds that there is a difference in his skeleton when he has to do this special job.

Up to the present, it has been the business of the surgeon, and especially of the pathologist, to make surgery and medicine the most thoroughly confused of all sciences. I do not think that anything can be more confused than medicine is, with a lot of conditions attached that you call pathology. Yet I do not think that anything could be more simple. You get a pathological condition just as in the drainage scheme of a house when it is stopped up. The pelvic colon becomes dilated, and you get a binding down of the bowel. This is to prevent the whole of the large bowel from filling up with solid feces, just as everything is done to make it more perfect for its labor. Everything of that kind, however, tends to shorten life. The coal heaver, who carries a couple of hundred weight on his back is able to do so simply because he locks the joints between the spine and the ribs and between the ribs and the sternum, and makes of his skeleton simply a shelf on which two hundred pounds is put. Until he is old, he can do work which a young man, without that change in his skeleton, could not do. But although that change makes him a splendid coal heaver, he can breathe only with his abdomen. His chest is fixed. If he gets an attack of bronchitis, he cannot breathe with his chest and dies very soon. The condition that develops and forms the obstruction that I have called the first kink, there, because it is the first developed and the last of the drainage scheme, is, at first, a good thing, but after a time, it becomes an obstruction. At that point, sooner or later, you get changes that are ulcerative, inflammatory or cancerous. Later, you get ulceration in the splenic flexure and the cecum. It thrusts the heavy colon into the pelvis. It develops what I call crystallization of the lines of force.

People are inclined to say that these things are congenital. I have opened heaps of infants, and have never seen a child with the slightest evidence of a kink. They are folds of peritoneum. If one thinks the condition is congenital, it is because he does not know. I think that these things are all developed, just as the changes in the coal heaver are. You have a big, loaded cecum, which tends to drop into the pelvis. You get lines of

crystallization, which develop outside and form a band, or membrane. On the inside, there forms the inner ligament of the cecum; and under that surface you get lines of crystallization formed. But that differs in no way from the last kink of any acquired band. You call the one on the outside, Jackson's membrane; and that on the inside, Lane's kink.

I have never seen anyone in a hospital on this side of the water try to treat this condition. They will try to do a gastro-enterostomy. The question with them is not, "Why do you do an operation?" but "How do you do it?" I have talked for a whole hour to audiences; and at the end, someone would say, "What sutures do you use?" or "what is the operation?" These different operations are done to meet certain conditions. You have the cecum dropping into the pelvis and developing either an appendix condition, which anchors it up, or a band called a Lane's kink.

As long as this delay is limited to the big bowel, little or no harm results. The evils of constipation related to it are of very little primary importance. But when it involves delay in the small intestine, you get two series of changes. The moment that the effluent current is delayed by an appendix, which hitches up the bowel, or a kink, you get two series of changes: changes due to the presence of stasis.

Before we go further, it is well to remember that mechanical changes differ in the two sexes. Women get one disease, and men another. What you want to know is why? The mechanics of the woman is different from that of the man, and that of the young woman from that of one who has carried children. Everything that occurs in nature is simple, if we have the sense to get to the ground of it. Everything is purely mechanical. Nothing comes accidentally. It follows the simple mechanical laws.

Let us follow the mechanical processes in the small intestine. In a taut bowel, the pull of the small intestine on the end of the duodenum obstructs the small intestine at that point. The obstruction results in its distention. The material cannot get by, and cannot return to the stomach, because the pylorus is shut; and from there you get a loop, part of which is free, and the rest controlled. If you were to take the duodenum and blow it up, that part would become overdilated, and later, congested. It then becomes restricted as regards its movement, and later still, abraded and ulcerated in its upper part. Why do ulcers

occur in only one place? All are exposed to the same mechanical conditions. The reason that we get changes in the first part is that it is the portion that becomes most distended and is exposed to the greatest pressure, because it is unprotected. The material cannot get back into the stomach, because of the spasmodic action of the pylorus; and you have early changes in the duodenum, and these are infinitely more frequent than we have supposed. Later, the spasm results in hypertrophy and dilatation of the stomach.

From this you get two mechanical conditions: the forcible impact of the contents of the stomach against the pylorus, and a tearing strain against the lesser curvature. From the tearing strain on the lesser curvature, you get an ulcer; and the surgeon says, when he gets an ulcer, "What operation do you perform?" and "What is the best way to do it?" He does not go into causes; and therefore, his treatment is not sound. Simple ulcer comes where you would expect to find it, on the lesser curvature. Twenty-five per cent. of impacted ulcers occur at the pylorus. In cancer, the larger proportion of the cancerous ulcers are at the pylorus, and not on the lesser curvature. Then you get the stomach full and get cardiospasm, because the esophagus is, naturally, reluctant to let the contents of the stomach pass into it.

The conditions in the duodenum are much more common than we suppose, and the reasons for this are simple. The minute you get spasm of the pylorus, you destroy conditions that are demonstrable in the duodenum. The X-ray man says that he cannot get pictures there, because the stomach has not the power to drive its contents onward. The conditions are very curable, when not sufficiently prolonged to produce cancer by impact. One of the most difficult things is to get cancer in the duodenum.

These are some of the things that result from stasis, but there is a vastly more far-reaching thing. Our bacteriologist has worked it out thoroughly, and I think that he has shown that every ten minutes to half an hour makes a marked difference in the contents of the small intestine, if infected by organisms. People may say, "That takes only two or three hours more," as if that made no difference. It makes a great difference. The organisms multiply rapidly, and ascend through the liver, the pancreas, and even the stomach, and mix with those from the bowel. In consequence of these infections of the biliary ducts, you get gall stones;

and you get the liver exposed to attacks in three separate ways. You have the branches of the biliary ducts extending among the liver cells. You have the liver supplied with blood from a small intestine whose contents are foul; and the hepatic artery, by material passed by an imperfect process of filtration from the general system. The supply of infection extends up along the ducts, and foul matter from the portal vein goes into the blood from the hepatic artery.

That applies to every organ in the body. The liver helps in dealing with the contents of the small intestine, which are normally sterile. When it has a lot of filth thrown into it, a great deal of this gets through and enters every tissue of the body. If you want to see the effect of auto-intoxication, take a dark-haired woman who has started life with round breasts and a good shape, and you will find that her breasts hang down, she becomes limp, her appetite for food is lost, and she loses her sexual desire. This is one of the greatest sources of misery.

I will tell you of a lady whose husband brought her to see me. She had led a happy life. When young, she had had plenty of sexual appetite and desire; but she had lost it all. His medical adviser asked me to remove her big intestine. I did so, and converted her into a perfectly healthy woman. She sent her love to me, and said that if all women had their big intestine removed, there would be no necessity for a divorce court.

I am not going to tire you by talking too long; but I want to say that the whole point of this is that I believe that all these operations on the intestine are performed with a single object, to overcome the infection that exists, and that the most perfect and simple way to do this is to remove the large intestine. The partial removal, which I tried and dropped because I found it absurd, is of no avail; because you still have the last kink. Sooner or later, you will learn to treat these conditions, either by vaccination or some form of dietetics, and thus obviate a great deal of the trouble.

I might cite a case to show the value of vaccine: A man was sent home from South Africa. He had had arteritis obliterans, and had had his leg removed. He was not expected to arrive home alive, because his other leg was becoming gangrenous. I found a big ulcer on his stump. He had gangrene of the toe, and was taking large doses of morphia. I went home and said

nothing. I said to myself, "That man has pyorrhea and other evidences of stasis; so before doing anything, I will get our bacteriologist to go over his feces and gums." This was done, and he found in the feces a *Bacillus coli* of a peculiar nature. He examined the organisms in the gums also, and used those with a double culture. The *Bacillus coli* vaccine produced a remarkable reaction, and within two months the man left the hospital, apparently perfectly well. The organisms from the gums produced no effect. The case is interesting. I got Sir James Mackenzie to see the case with me. What he said supported my view of angina pectoris: that the pain is due to the contraction of a muscle that is badly nourished, owing to constriction of the coronary artery. The patient has gone back to St. A. to study. I got Sir Amrath Wright to see him. We are now treating another case of that kind in that way. I got another case, and examination of the feces shows that it contained the same organisms, and the reaction to the vaccine was the same.

I might talk for hours, but I do not think that I shall say anything more.



